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HYPOTHERMIA IN CARDIAC SURGERY.[1]

By VICTOR HERCUS, M.B., B.S., M.R.C.P. (Edinburgh), D.A. (London),

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HYPOTHERMIA, when it is well controlled (Cohen and Hercus, 1959), is a safe procedure and occupies an important place in the field of cardiac surgery.

Some of the physiological problems associated with lowered body temperature have been investigated.

Temperature Gradients.

The temperature of the cerebral cortex during cooling and rewarming was measured in sheep (Hercus *et alii, 1959*). *This was done by using a needle with a thermistor* at its tip, placed through a drill hole, made at the base of a horn, directly into the cerebral cortex, the location of the needle having been determined previously in a bisected *sheep's skull. Simultaneous measurements were made in* the œsophagus (lower third), the central blood stream (right atrium), the rectum, the naso-pharynx and the subcutaneous tissues. It was found that the œsophageal, brain and central blood-stream temperatures were very *closely related during surface cooling, stabilization and* rewarming. The rectum and naso-pharynx were sluggish in reflecting changes in brain temperature and could be *quite misleading; the subcutaneous temperature readings* were at great variance with those from the other sites.

Accurate recording of the œsophageal temperature reflects the cerebral temperature, and the circulation can be interrupted with confidence, the temperature of the brain at the time being precisely known.

Metabolic Changes.

The metabolism of the body is reduced during hypothermia, different organs and parts of the body varying *considerably. The brain has one of the greatest reductions* in oxygen consumption (Rosomoff, 1956) and the heart one of the least. The arterio-venous oxygen saturation difference between coronary artery and coronary sinus

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959. Part of this work was supported by a grant from The Royal Australasian College of Physicians.

blood is increased as cooling proceeds (Penrod, 1951), and the heart performs relatively greater work for less coronary blood flow than at normal body temperatures (*McMillan, 1956*).

Intracellular Changes in Muscle.

The effects produced by this reduction in metabolism have been little studied in the cells themselves. Knocker (1955) and Fisher (1956) showed deleterious effects on the intact liver and on liver slices as a result of hypothermia. We have studied the changes occurring in skeletal muscle of sheep in respect of their sodium, potassium and water content before cooling, when cooled, in an iced-water bath to 28°–30° C., and after rewarming of the whole animal (Hercus and Bowman, 1959). The extracellular space of the muscle was determined by the use of radioactive sulphate ($S^{35}O_4$). Serum sampling and muscle biopsies permitted determinations of serum sodium and potassium contents, hæmatocrit, and muscle sodium, potassium and water contents. The results of these studies were as follows. (i) There was a fall in the serum potassium level, yet hæmoconcentration developed, resulting in a rise in the hæmatocrit. (ii) Muscle cells were found to have an increase in their intracellular water, total sodium and sodium concentrations; the potassium in the cells was diluted. (iii) The sulphate space of muscle was reduced.

These changes suggest either an anoxic effect, possibly resulting from the hæmodynamic changes (lowered cardiac output, increased blood viscosity, lowered blood pressure, lowered heart rate and peripheral vasoconstriction) produced by hypothermia, or a direct depression either of cell metabolism, reducing its energy production, or of some enzyme system necessary to maintain an effective "sodium pump".

The important feature of these changes was their reversibility on rewarming of the subject, with no subsequent deleterious effects. This is borne out clinically, in that patients recover with no muscle weakness, which would be expected if changes like these persisted in the muscle cells.

Coronary Perfusion.

Coronary perfusion, we believe, is of great importance in all cases in which the circulatory arrest lasts for more than three to four minutes. Perfusion of fresh heparinized oxygenated blood (Cohen, 1958) at 35° C. at the rate of 3 ml. per kilogram of body weight per minute into the coronary arteries supplies the heart muscle with oxygen and prevents the harmful effects of anoxia, known to occur with great rapidity (Hercus *et alii*, 1955). This results in strong and vigorous contractions of the heart as soon as the circulation is restored. Ventricular fibrillation is thus averted, and a normal cardiac output is achieved forthwith. The use of coronary perfusion is more satisfactory, safe and physiological than the alternatives of doing nothing at all (letting the heart become anoxic and expecting it to function normally on restoration of the circulation), or using chemicals to induce cardiac arrest and relying on massage to remove them when the clamps occluding the circulation are removed. Coronary perfusion is a safe and simple procedure, and essential if the heart is to be respected.

The Future of Hypothermia in Cardiac Surgery.

We propose to continue using hypothermia for all patients with pulmonary valvular stenosis or coarctation of the aorta, for "poor risk" patients with Fallot's tetralogy and atrial septal defects (secundum), until such time as the risk of by-pass techniques becomes the same as hypothermia methods which, at the present time, involve practically no risk at all.

Moderate hypothermia with by-pass is being utilized in many centres in the United States of America, but this is done to give extra protection to the patient, because of inadequate blood oxygenation at high flow rates or with inadequate pumps to give effective flows. When machines are improved, no doubt this type of hypothermia will cease.

Deep hypothermia has been used in places such as Duke (*by Sealy*) and *Westminster (by Drew), but results are* difficult to evaluate; indications are that they offer no improvement over existing methods. Much animal experimental work is being conducted with deep hypothermia, but many unexplained deaths occur.

As Bigelow stated in 1954: "We must know a lot more concerning oxygen transfer from blood to the tissues and nerve conduction at low body temperatures." He is actively engaged in studying the properties and role of the hibernating gland obtained from such mammals.

Cardiac arrest by perfusion of the heart with cold oxygenated blood is being used in certain centres. This is achieved by tapping the arterial line of a pump oxygenator and passing the blood through a coil immersed in iced water. We have seen hearts start performing actively and *effectively after over two hours of such arrest.*

The future of hypothermia in heart surgery is still *full of interest and possibilities, and is dependent more* and more upon knowledge of the fundamental processes involved in cell metabolism.

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HYPOTHERMIA FOR CARDIAC SURGERY IN CHILDREN.[1]

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FOR cardiac surgery in children, when hypothermia is utilized, various technical requirements are desirable, the essential elements of which are as follows: (i) A central body temperature which can be maintained accurately at any desired level, usually 30° C. This is estimated by the temperature recorded in the lower third of the œsophagus (Cooper and Kenyon, 1957). (ii) A sufficiently rapid rate of change in body temperature to avoid any extension of the normal operating time. (iii) Means whereby the body temperature may be conveniently raised or lowered at any time during the operation. (iv) Restoration of the body temperature to normal as soon as possible after the completion of the operation, with minimal handling of the patient. (v) Accurate control of the rate of change of body temperature, and facilities to stabilize it rapidly at any desired level, thus preventing any "overswing".

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

(vi) The avoidance of any additional major surgery solely for the purpose of producing cooling.

The use of blood-stream "extra-corporeal" cooling fulfils most of these requirements, but has certain disadvantages which will be discussed later (Delorme, 1952; Ross, 1954; Lucas, 1956).

Present methods of surface cooling, with or without the use of long-acting drugs, do not fulfil these requirements. The use of intragastric cooling (Khalil, 1957) is slow, and *is not a practical proposition in association with surgery* (Bigelow, 1950; Swan, 1953; Lewis, 1953; Bailey, 1954).

The method presented here relies on body surface action. The skin of the patient is in close contact with rubber blankets containing a continuous capillary, through which *hot, cold or tepid water is recirculated.*

The machine[1] used to supply and pump the water *contains three tanks filled with water at 40°–42° C.,* 25°–28° C. and 2°–3° C. respectively. Outflow and inflow connexions for five blankets are housed below a central control panel, which regulates the rate of flow and the contents of each blanket (Figure I). Automatic valves

FIGURE I.

The patient is shown on the operating table with polythene-covered rubber blankets tied in close contact with the body. In the background is the "hypothermia *machine", and on its top left-hand corner is the* electrical thermometer.

ensure non-mixing between tanks when the water circulating in the blankets is changed. Hence it is possible to circulate water from any tank through any blanket at whatever rate is desired. The machine is readily moved to a convenient position in the operating theatre, and may be kept at any desired distance from the patient. All connexions are of a simple bayonet, self-sealing type.

The blankets[2] are made of rubber, and contain a continuous capillary, enabling water to be circulated through their entirety. Three blankets measuring 18 by 40 in., 12 by 32 in. and 8 by 32 in. have been found adequate for all children, from small babies to children aged 13 years.

It is essential to have available facilities for accurately and quickly recording the body temperature, preferably in more than one situation. The recording apparatus should be situated conveniently close to the hypothermia machine and well away from the patient, so that no inconvenience will be caused to surgeon or anæsthetist during the operation.

Method.

The anæsthetized, unclothed patient is placed on the operating table on a large rubber blanket. All necessary leads are then connected. It is considered essential to monitor the heart's action continuously, using a cathode ray oscilloscope, whenever hypothermia is employed. In all *"open heart" procedures, a continuous record of the intra-*arterial blood pressure is also considered desirable.

The child is placed in position according to the nature of the operation, and a narrow blanket is placed between the legs and thighs.

A third blanket is then placed on top of the patient, covering the abdomen and legs, and all blankets are then tied together, overlapping where necessary, but obtaining *maximum contact with the skin. The only exposed parts of* the body are the head, the neck, the arms and that part of the thorax where the surgeon is to operate (Figure I).

A long piece of half-inch rubber tubing is then coiled around the neck, fitting snugly but not too tightly, and making as many coils as possible (usually three or four) in order to obtain maximum skin contact (Figure II). This

FIGURE II.

The neck coil is shown in position. The chest is exposed for bilateral thoracotomy.

tubing makes the fourth connexion with the machine, one *spare connexion being left.*

Water at 2°–4° C. is circulated through all the blankets and the neck coil. The rectal and œsophageal temperatures are recorded at regular intervals in graph form, and so long as no alteration is made to the rate of flow through the blankets or the temperature of the water contained therein, it is found that there is a linear rate of fall in the body temperature. The precise time can be predicted in which *the body will cool to any desired temperature. As the body* temperature approaches the required level, it is possible to circulate tepid or hot water through any or all of the circuits, or any blanket can have its circulation stopped independently of the others. This allows great flexibility and ideal control and, of course, eliminates "overswing" entirely.

As soon as the blankets have been connected and the *patient is in position, the surgeon can scrub up and proceed* with the operation; then, while the operation continues, the temperature is lowered to the required degree and, when this point is reached, may be stabilized and held there. As soon as desired, rewarming can be inititated by circulating hot water through the blankets, and by the time the operation is completed, the temperature of the patient is invariably raised sufficienly to allow safe transport from the theatre (Figure III).

The child is then taken to a recovery ward, together with the temperature recording apparatus, and placed in an oxygen tent. The temperature is regularly recorded at ten-minute intervals, and active steps are taken to prevent any rise in temperature above 37° C.

This method of hypothermia has been used on more than 50 children undergoing cardiac surgery and has proved extremely satisfactory in all cases, an adequate rate of cooling being invariably maintained.

The use of a coil of rubber tubing around the neck, bringing the circulating water into close proximity with the large vessels in this region, accentuates the rate of fall in temperature (Figure IV).

[1] Designed and manufactured by Ebsray Pumps Pty. Ltd., Sydney.

[2] Manufactured by Dunlop Rubber Australia Ltd., Sydney.

The average rate of fall in the six cases prior to the use of neck cooling was 4·5° C. per hour, and after neck cooling was introduced the average fall in temperature was increased to 5·5° C. per hour.

Infants and small children may be cooled at a more rapid rate than larger children, owing to their greater surface area in proportion to their weight. Other factors will influence the rate of cooling, such as a large shunt

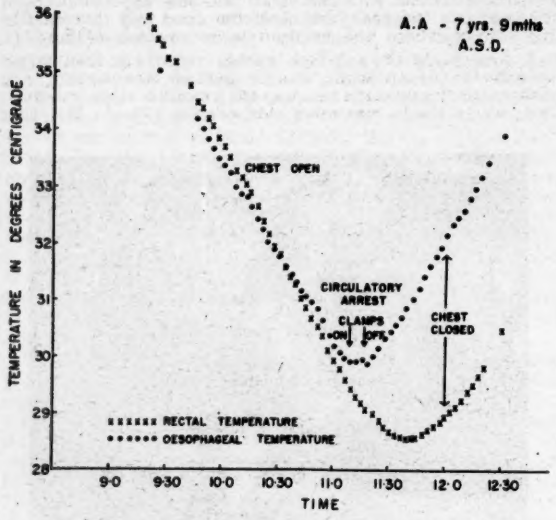

FIGURE III.

Illustrating the linear rate of fall and rewarming in a child with an uncomplicated atrial septal defect repaired under hypothermia. This graph demonstrates the accuracy with which the temperature may be controlled, and shows the sluggish manner in which the rectal recording reflects rapid changes in body temperature. The lead in the lower part of the œsophagus gives a much more accurate indication of the changes taking place.

in the circulation, a coarctation with a poor blood supply to the lower half of the body, or obesity in a patient with a poor peripheral circulation. Such factors will retard the rate of cooling, not only by this method but also by blood-stream cooling (Lucas, 1958).

Rewarming has presented no difficulties; but temporary setbacks may occur if large amounts of cold blood are quickly tranfused. All blood is now warmed to 35° C. prior to transfusion, and this problem has been eliminated. It is possible to rewarm the patient when the only effective circulation is cardiac massage.

No cardiac irregularities have occurred in this series that could reasonably be attributed to the use of hypothermia.

Coronary Perfusion as an Adjunct to Hypothermia.

When it is expected that it will be necessary to maintain inflow occlusion for a period in excess of three or four minutes, it is considered advisable to maintain adequate oxygenation of the myocardium by the perfusion of the coronary arteries with oxygenated heparinized blood.

If this is not done, it will be found that an appreciable proportion of patients will enter ventricular fibrillation, either during the intracardiac procedure or immediately after reestablishment of the circulation (Swan, 1953, quoted by Maloney *et alii*, 1957). In other cases a satisfactory ventricular beat is slow to return and the systemic arterial pressure is slow to return to normal, so that the cerebral circulation may be inadequate for some time after the period of inflow occlusion. Fibrillation, when it occurs, is always worrying in the cold heart and may be difficult to reverse.

If the myocardium is perfused with oxygenated blood at a rate in excess of 3 ml. per kilogram per minute during inflow occlusion, it will be found that major irregularities of rhythm can be virtually abolished, and the systemic arterial pressure will return to normal within 10 to 15 seconds of release of the caval tourniquets (Riberi *et alii*, 1956).

The preparation of arterialized blood is extremely simple, and has recently been described (Cohen, 1958). It is introduced into the coronary circulation by an 18 gauge needle, which is inserted into the aorta through a special holder sutured to the aorta prior to arrest of the circulation. After the venæ cavæ are occluded, the heart is allowed to empty, after which the aorta is clamped and perfusion is commenced.

Since introducing this technique, we have not had ventricular fibrillation occur in any of our cases.

Some surgeons hold that they can adequately protect the myocardium by inducing cardiac arrest with potassium or acetylcholine during the period of inflow occlusion; but we believe that any form of cardiac arrest is inadvisable without the added safeguard of an extracorporeal circulation, to ensure that a satisfactory cardiac action may be safely reestablished in due course.

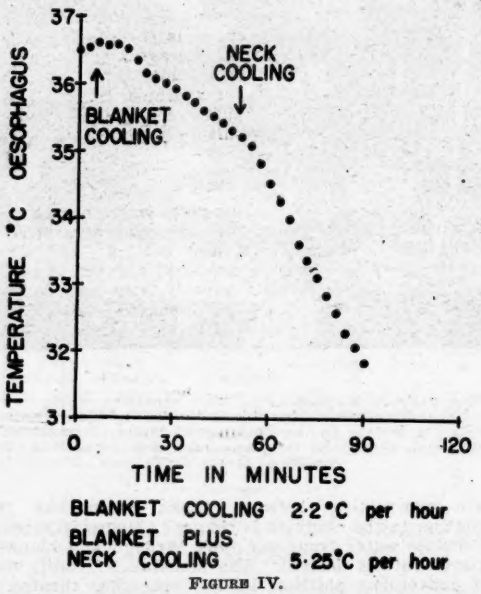

FIGURE IV.

Illustrating the value of the neck coil in accelerating cooling. Blanket cooling only was used for the first 45 minutes, after which is was supplemented with the neck coil. The marked increase in the rate of cooling can be seen. Rewarming is also facilitated in a similar manner.

Selection of Patients for Hypothermia.

Atrial Septal Defects.

Atrial septal defects of secundum or sinus venosus types are eminently suitable for closure under hypothermia. It is permissible to utilize a period of ten minutes' inflow occlusion for this purpose and this will in general prove adequate. Although it has not proved to be necessary in any of our cases, we would not hesitate to reestablish adequate cardiac action and then make use of a second period of inflow occlusion should the need ever arise.

Septum primum defects are not considered suitable for closure under hypothermia.

Pulmonary Stenosis.

Valvar pulmonary stenosis may be dealt with very satisfactorily under hypothermia, and it is rarely neces-

sary to arrest the circulation for more than two to three minutes to achieve this. Isolated infundibular stenosis is more safely dealt with by means of a cardio-pulmonary by-pass.

Aortic Stenosis.

Aortic stenosis may be dealt with under hypothermia, and this has been done satisfactorily in our unit. However, since the aortic valve is such a critical structure, we believe that it is preferable to utilize cardio-pulmonary by-pass in these cases, and this will be our policy in the future.

Coarctation of the Aorta.

We hold strongly that all child patients with coarctation undergoing surgery are best operated on under hypothermia. In one case of uncomplicated resection of a coarctation in our unit, paraplegia, fortunately incomplete, developed. Since then, all such patients have been operated on with the added protection of hypothermia, and we have been pleasantly surprised by the way in which it has simplified the operative procedure and by the smooth post-operative course of such patients.

Tetralogy of Fallot.

In the surgical treatment of severely limited cyanotic children, particularly in infancy, it is considered wise to employ hypothermia, since it is necessary to clamp one pulmonary artery for an appreciable time while an anastomotic procedure is being performed. It is thought that hypothermia in such cases reduces the risk of permanent cerebral damage resulting from anoxia during the operative procedure.

Conclusion.

Hypothermia and total cardio-pulmonary by-pass are playing an important part in the correction of a steadily increasing number of congenital heart defects. The manner in which these two techniques are used and the extent to which they are employed vary greatly from unit to unit, and in any one unit changes are constantly being made, as one would expect in a field that has been so recently developed (Brock, 1956; Delorme, 1956; Ross, 1956).

It is our firm belief that hypothermia, particularly with the use of the technique that has been described, is the procedure of choice for the correction of certain abnormalities, as has been detailed in the text above, and that, used in this manner, it provides a safe and effective method for the cure of a number of important congenital heart defects.

Summary.

1. A method of hypothermia and rewarming relying on body surface action is described in detail.

2. The use of coronary perfusion of oxygenated blood as an adjunct to hypothermia in open heart surgery is described.

3. The selection of patients for operation under hypothermia is discussed briefly.

Acknowledgements.

Our thanks are due to Dunlop Rubber Australia Ltd., for donating the special blankets used in this procedure, and in particular to Mr. G. Waring and Mr. J. F. Sharpe of that firm for the time and trouble involved in developing a satisfactory set of blankets for our requirements.

In particular we pay tribute to Mr. V. R. Ebsary, whose ingenuity and initiative were responsible for the development of the hypothermia equipment. This ingeniously contrived machine has done all that has been required of it in every case in which it has been used. In addition, Mr. Ebsary has attended, in every case in which hypothermia has been employed, to operate the machine for us. Although it is simple to use, his skilful regulation of the body temperature has relieved the anæsthetic and surgical staff from any worries in this regard, and has allowed us to concentrate wholly on the medical aspects of the particular case.

Finally, we recognize the contribution of Mr. A. Harper, of the Commonwealth Scientific and Industrial Research Organization, who also gave willingly of his time and knowledge to advise us on certain technical aspects of heat transference, and who kindly attended the operating theatre for the first few cases to assist in the organization and supervision of this procedure.

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TECHNIQUES FOR OXYGEN THERAPY IN INFANCY AND CHILDHOOD.[1]

By R. GODFREY,
Perth.

IF deaths from congenital abnormalities at Princess Margaret Hospital, Perth, are compared with those from infection, a striking variation is revealed (Figure I). Treatment has unfortunately not yet greatly influenced the number of deaths from congenital abnormalities, and if these figures are placed next to the figures for infection, they demonstrate, even more strikingly than would admission or population indices, how great the reduction has been in the number of deaths from infection between 1953 and 1958.

This reduction has been progressively attained in the intervening years, and approximately half of it has

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

occurred in deaths from respiratory infections. No new antibiotics of any significance have been introduced during this period, and I believe the reduction is due to general improvement in patient care.

Oxygen therapy makes an important contribution to the management of respiratory infections, and our methods have recently been reviewed. The purpose of this paper is to discuss equipment and techniques for oxygen therapy in childhood, and to detail certain equipment modifications that have improved the efficiency of treatment.

The limitations and dangers of oxygen treatment of new-born and premature infants are well known. These infants receive oxygen in rigid "incubators" that usually perform according to specification, and these will not be further discussed.

Shortly after this talk was accepted, an article by Batson and Young (1958) appeared reviewing the subject so thoroughly as to seem to make this paper redundant; but techniques of oxygen administration in other Australian pædiatric centres have recently been observed, and the following remarks may still be relevant.

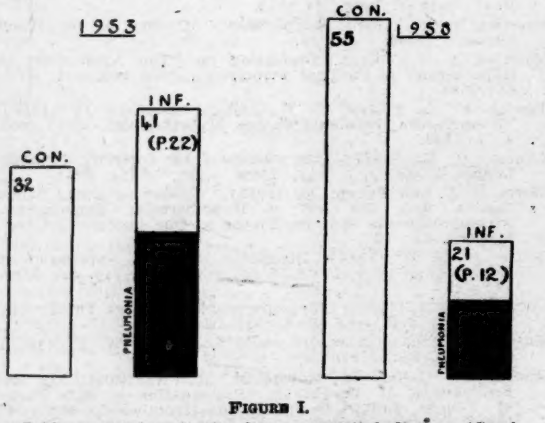

FIGURE I.

Table comparing deaths from congenital disease (Con.) and infectious disease (Inf.) in 1953 with those in 1958. Section in black (P), pneumonia.

Oxygen may be administered by nasal catheter, oxygen mask or tent. Nasal catheters so distress infants that they are almost useless, and masks, though capable of delivering high oxygen concentrations in older children, upset even the most cooperative and produce a sense of claustrophobia, so that except for a short time in an emergency nearly all infants and children requiring oxygen receive it in some form of tent.

An article by the Committee of Public Health Relations of the New York Academy of Medicine in 1950 discussed the use of oxygen tents and recommended a concentration of 50 volumes *per centum* at a temperature of 15° to 20° C. and a relative humidity of 40% to 60%, stating that this could be achieved in a standard oxygen tent with a maintenance flow of 10 to 12 litres per minute.

Batson and Young, however, have found that the concentration actually achieved in tents varies from time to time, according to the care with which the canopy is applied and the amount of interference. In their experience a concentration of 50% was not often attained, and Morrison (1959), treating patients with severe bronchiolitis, encountered the same difficulties.

Batson and Young recommend a rigid "Lucite" box containing an ice compartment, closed at the bottom except for a semicircular aperture through which the patient's legs pass. A plastic sheet attached to the margins of the semicircular aperture can be fitted tightly round the patient's hips, to prevent leakage of oxygen through the aperture. This box is open at the top, and these authors claim that cooled oxygen, being denser than air, forms a layer at the bottom as it enters the box, and that diffusion is sufficiently slow to permit high concentrations at the level of the patient's nose with low rates of oxygen flow.

Morrison recommends the use of a small, rigid "Perspex" box placed over the baby, with an attached plastic apron that can be tucked underneath to provide the high concentrations sometimes necessary for treating babies with severe asphyxia. With her equipment, concentrations as high as 90% were achieved with a flow of only 5 litres per minute. The box is closed at the top, and is cooled by circulating the oxygen over a compartment containing ice placed under the cot.

For some years we have used a small rigid "Perspex" box, closed at the top, that rests on the bed, with an aperture at the front for the patient's legs. However, it has been found, that despite the comparatively small capacity, it was not possible to achieve a concentration of 80% to 90% in this box without very wasteful use of oxygen. By applying an ensheathing layer of plastic completely covering the bottom of the box and drawn up round its sides and end like a sock (Figure II), the

FIGURE II.

"Perspex" oxygen box showing plastic "sock".

efficiency of the apparatus has been greatly improved. High concentrations of oxygen can now easily be achieved.

Figure III shows diagrammatically the construction of this box. The oxygen enters the metal ice chamber through a T-shaped perforated tube, and is cooled. It then passes out through the holes at the top of the ice chamber and, being of relatively high density, sinks to reach the infant. The metal ice chamber is removable, can be entered from the top for renewal of ice and provides a large conducting surface.

A metal tray is placed under the ice chamber to collect the condensate which drips off the cold surface. It is connected to a tube draining into a receptacle by the bedside. The box can be kept at temperatures below 75° F. with babies weighing up to 15 lb. A larger box would be suitable for bigger infants. Carbon dioxide tension does not rise above 0·3%, and tests of the relative humidity have averaged 60%.

For older children we have used a "Croupette"[1] as an oxygen tent; but although it is excellent as a moisture-providing canopy, it has all the already-mentioned faults of the standard oxygen tent, and the added defect of imperfect cooling producing a sense of suffocation in the patient. For this reason our "Croupettes" have recently been altered to provide a higher oxygen concentration and more efficient cooling.

The canopy has been modified by joining its lower margins together with an added section of plastic sheet

[1] Air Shields Company, Halboro, P.A.

passing beneath the patient. A zipper was then inserted in its upper section, allowing it to be opened at the top. It is used as follows. With the zipper completely released, the canopy is first laid upon the bed like a sheet. A section of "Estafoam" sponge plastic and a draw-sheet are then placed upon it, and the "Croupette" and patient are placed in position. The canopy is then drawn up and round the "Croupette's" frame as if wrapping a parcel. It is then attached to the margins of the "Croupette" at the head of the bed in the usual

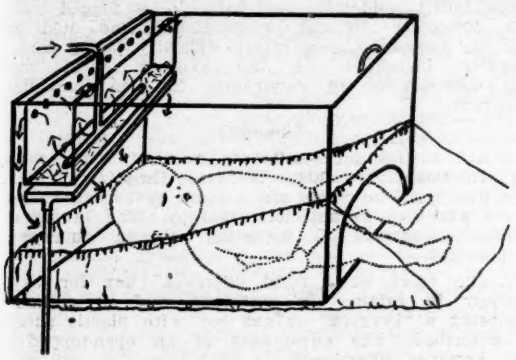

FIGURE III.
Diagrammatic representation of Figure II.

way, and the zipper is closed at the top, making a continuous encircling sleeve above and below the patient, the only opening of which is at the end facing the foot of the bed. If the child is large, this aperture can be closed round the hips by a tape; but if he is small it can be completely closed off by spring clips (Figure IV).

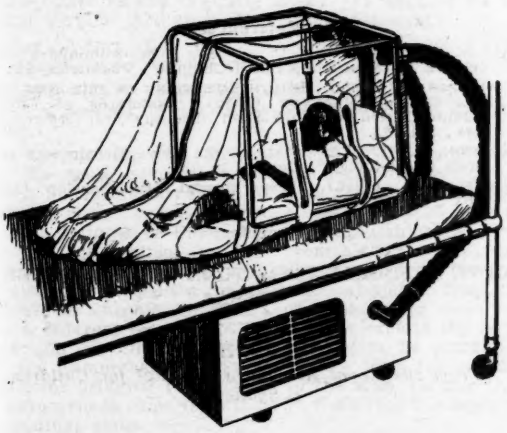

FIGURE IV.
Modified "Croupette", showing method of application of canopy with zipper fastener at the top and refrigerator compartment.

Since the canopy is now continuous above, around and beneath the patient, there is no need to "tuck it in", and oxygen loss from leakage round the margins of the canopy is eliminiated. Diffusion is nonetheless sufficiently rapid to prevent accumulation of carbon dioxide.

The releasing zipper makes it possible to enter the canopy from the top and to apply it to very ill patients by rolling it under the recumbent child, in the manner commonly used by nurses for changing sheets.

Temperature is controlled by circulating the tent oxygen through a refrigeration compartment specially designed for the purpose. The refrigerator is on wheels, fits neatly under the bed and can be easily moved. A throttle valve controls the rate of circulation and hence the temperature in the tent.

A concentration of 80% oxygen can be easily maintained, although with older children it may be necessary to use more than 15 litres per minute to achieve this. A point of practical importance is that not more than 8 litres per minute can be passed through the fine nozzle of the "Croupette's" vaporizer, and attempts to increase the flow only result in "blowing-off" of oxygen at connexions or through the safety valve. If additional oxygen is required, it must be delivered to the tent through a separate opening.

Discussion.

At the Princess Margaret Hospital, all sick infants are nursed in an air-conditioned ward; but elsewhere the cooling capacity of a "Perspex" box of the type used by us for infants may possibly be inadequate in very hot weather. In such conditions the open-topped box would seem to have obvious advantages, and it was therefore thought worthy of trial.

In an attempt to test its value, one of our boxes was modified to resemble that described by Batson and Young. The sides were raised to 20 inches, the ice chamber was filled with ice, and measurements of oxygen concentration were made. It did not prove as satisfactory as was claimed. Figure V compares results

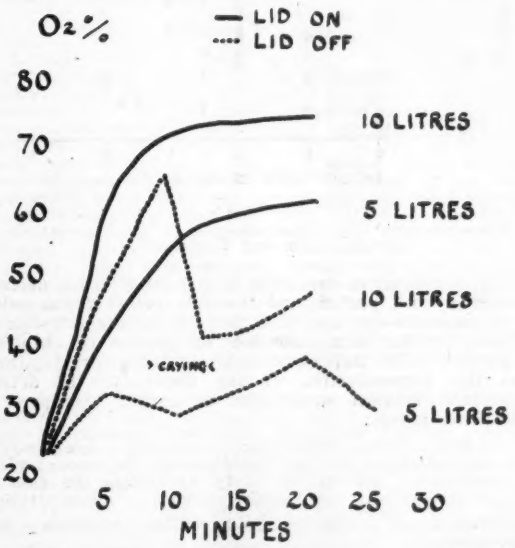

FIGURE V.
Graph of oxygen concentrations obtained in high "Perspex" box at 5 litres and 10 litres per minute, with top (continuous line) and without (broken line).

obtained at different rates of oxygen flow with and without a lid. With the lid on, oxygen concentration rose in a smooth curve, and approximately the same curve was achieved with the lid off, provided the air was still. However, any disturbance, even the activity of the baby crying, caused sufficient turbulence to spill the oxygen and immediately lower the concentration. After repeated tests we were reluctantly forced to the conclusion that even when a high-sided box is used, some form of cover is still necessary to prevent air currents from sweeping oxygen out.

Actually, a study of Figure VI, taken from Batson and Young's article, shows that even in their hands

concentrations achieved varied widely. For this reason it is difficult to understand their denial of the need for enclosing the top of their oxygen box.

There is no doubt that cooled oxygen does form a layer if it is not disturbed, and readings taken at different levels in a box showed a progressive increase in oxygen concentration, the lower the sample is taken. This does suggest that only a draught-excluding cover is required.

The rapid collection of oxygen at low levels is, of course, therapeutically useful, and the character of the curve in Figure IV indicates that the concentration of oxygen at the level of the baby's nose will rise a very few minutes after the box has been opened for nursing attention.

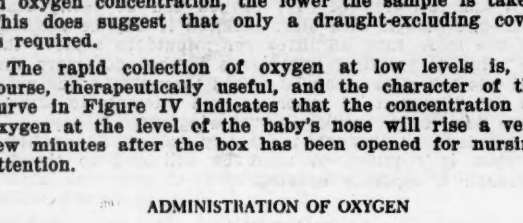

FIGURE VI.

Concentrations of oxygen achieved in open-topped box (from Batson and Young, 1958).

The modifications described here have all been derived from the work quoted, and the only point of discussion is to question the assertion that an open-topped box is satisfactory for administration of oxygen to children. A possibly more important result of these investigations was the demonstration of the importance of active, interested, medical supervision of oxygen therapy. To quote one report:

> Still many physicians responsible for oxygen administration are not familiar with the amount to be prescribed, nor do they fully appreciate the dangers associated with the various methods of administration.

Experience in this hospital fully endorses this statement.

The following points require emphasis.

1. Regular checks of connexions, rate of oxygen flow and application of the canopy are absolutely essential.

2. Some form of simply used oxygen analyser should always be available in the wards.[1] Twice-daily measurements have been recommended; but whenever the condition of a child in oxygen deteriorates, a measurement should be made immediately.

3. A thermometer should be in the tent or box at all times, and regular readings (for example, hourly) should be made. Comfort inside a tent depends greatly on the temperature. For febrile small infants a temperature of 75° to 78° F. appears to be comfortable, but for older children it should probably be less.

4. Oxygen equipment should, for most purposes, produce a humidity of at least 60%. In acute laryngeal stridor up to 100% may be needed. A hand-aspirated psychrometer[1] can conveniently be used to check the relative humidity achieved by the equipment.

5. Printed, detailed instructions for its use should be attached to all oxygen equipment. Instructions prepared for our own equipment include graphs to show how fast oxygen concentrate rises at different rates of flow.

6. All children requiring oxygen should be nursed with the chest exposed. In our recent experience of a severe bronchiolitis epidemic, one baby in a fogged oxygen box developed bilateral pneumothorax and died without his increasing respiratory difficulty being detected. Regular inspection of the exposed chest makes any deterioration of respiratory function immediately apparent.

[1] Oxygen concentrations were measured by a "Bechmann" analyser. An alternative is the "Mira" analyser.

Summary.

1. A reduction is recorded in mortality from respiratory infections at Princess Margaret Hospital during the last five years—a period when there have been no significant advances in antibiotic therapy. The influence of general management, including oxygen therapy is emphasized.

2. The value of a rigid "Perspex" box for oxygen therapy in infancy is confirmed, and a method of enclosing a "Perspex" oxygen boy with plastic sheeting is described. The advantages of an open-topped box are, however, questioned.

3. Modifications to a "Croupette" are described that provide more efficient cooling and minimize oxygen losses.

4. The importance of careful supervision of oxygen therapy, including temperature control and frequent checks by a simply-used oxygen analyser during actual treatment, is emphasized.

5. It is recommended that children in oxygen should be nursed with their chests exposed to facilitate observation of respiratory function, so that deterioration may be readily seen and its cause sought.

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ACUTE SUBDURAL HÆMATOMA IN THE POSTERIOR FOSSA.[2]

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SUBDURAL HÆMATOMA over the vertex has been recognized for many years, and the importance of this condition in infancy has been stressed in pædiatric literature over the last ten years. There have, however, been few cases reported in which the hæmatoma was found in the posterior fossa. This is understandable, because the majority of such cases are fatal at birth, and do not appear in clinical reports. The question as to whether hæmorrhage occurs in the posterior fossa or over the vertex may well be a matter of chance, and it would be useful to examine

[1] Humidity tests have been made with a hand-aspirated psychrometer, model HA2 (Bendix Aviation Corporation, Baltimore, Maryland).

[2] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

the various views that have been put forward to explain the source of hæmorrhage in these cases. Edith Potter (1952) points out that the intracranial cavity is the most common site of fatal or crippling injury in the neonate. Tears of the dural septa are due to the unequal stresses of labour. Forces equally distributed on both sides of the head are not likely to rupture either the falx or the tentorium; but if the force is asymmetrical, one leaf of the tentorium will be relaxed while the other is under excessive tension and may rupture. If a simple laceration of the margin occurs, no hæmorrhage results; but as the tentorium tears, the rent extends medially into the straight sinus or laterally into the transverse sinus, producing fatal hæmorrhage. Rupture of the vein of Galen is almost invariably fatal, large clots are found filling up the posterior fossa, and blood extends between the hemispheres and in a thin film over the surface of the brain.

Milroy Paul (1955), in discussing hæmorrhages due to head injuries, states that from his experience in autopsy material, tears in large veinous sinuses are unknown except in the new-born. He describes tears found in the dural septa, either at the free edge of the falx at its junction with the tentorium associated with rupture of the internal cerebral veins, or in the tentorial notch where the tentorium passes onto the superior surface of the petrous temporal. He attributes these injuries to head moulding, and describes the distortion of the vault by head moulding, which forces the falx upwards and pulls the tentorium off the upper surface of the cerebellum, disrupting the "bridging veins". These veins were described by Leary (1939), and were the subject of critical analysis by Paul in his autopsy findings. Paul found that the vessels traversing the subdural space followed a constant pattern, thus: (i) a close-set network of venules forming a mesentery on the superior border of each hemisphere suspending the cerebral hemispheres from the dura; (ii) a similar set of venules passing from the superior surface of the cerebellum to the lateral sinus immediately below the tentorium; (iii) veins passing from the frontal poles and temporal poles to the dura mater; (iv) solitary bridging venules over the outer surface of the cerebral hemispheres and cerebellar lobes, and the under surface of the temporal and occipital lobes; (v) venules on the medial surface just above the corpus callosum.

Other writers state that "bridging veins", apart from the sinuses, are found in only one-third of the population.

Schipke (1954) described tears of the tentorium disclosed at autopsy in association with hæmatoma over the vertex.

On the clinical side, Schreiber (1957) has emphasized that the acute hæmorrhage of the neonatal period producing subdural hæmatoma of the vertex has a rapid progress, characterized, in many cases, by such definite signs as fullness of the fontanelle, unilateral third-nerve paralysis, twitchings on the contralateral side and dramatic improvement by surgical evacuation of the clot, and that this presents a different clinical picture from the subacute or chronic hæmatoma of infancy and may well have a different mechanism. In order to explain the large proportion of unilateral cases in his series, he postulated that the hæmorrhage in his cases came from the rupture of inferior bridging veins, while in the more chronic type the hæmorrhage came from the veins entering the superior longitudinal sinus.

All the evidence points to head moulding as the essential factor of the acute type of subdural hæmatoma, and this mechanism has not been in dispute since Holland's description in 1922. In the later stages of labour, head moulding would produce the unilateral stress with excessive tension on one leaf of the tentorium postulated by Edith Potter, would explain the tears found by Paul, would explain the unilateral predominance noted by Schreiber whether the hæmorrhage comes from inferior bridging veins or from solitary veins over the surface of the hemisphere, and would explain a combination of posterior fossa hæmorrhage with hæmorrhage over the vertex as noted by Schipke. The distinction between acute hæmorrhage in the neonate and chronic hæmatoma of infancy has been made by various authors, but sometimes without a full realization of the implications. Thus Levy (1957) recognized one of his cases as being an acute hæmatoma, and for five days carried out the treatment by aspiration recommended by Ingraham for the hæmatomas of infancy. The head increased in size from 36 to 37 cm., and the taps became less productive. Finally the child became grey and stuporose with tense fontanelle, and surgical intervention was forced on the author, who evacuated the clots through a burr hole with a good result. Earlier intervention might have produced an even more satisfactory result. Schipke (1954) realized the importance of the distinction, and was able to cure two patients by aspiration alone; this is possible when the blood is fluid and has not formed a clot. If it is important to recognize this condition early and treat it energetically when the hæmatoma is over the vertex, it is much more important when blood collects around the cerebellum, because, if it is untreated, the extra mass in the confined space of the posterior fossa will rapidly lead to an obstructive hydrocephalus, with a degree of brain destruction that would preclude any useful intervention at a later stage.

Although the hæmorrhage is usually rapidly fatal when it fills the posterior fossa, one should be influenced not by the gloomy statements that emanate from the study of autopsy material, but rather by the irrepressible optimism of the clinician: "So far there is no treatment for posterior fossa clot. It could be evacuated through the occipital bone." (Milroy Paul.) It is refreshing to know that as long ago as 1940 (Coblenz), a case was reported which is almost identical with the one to be presented here, the baby being cured by evacuation of the clot, with the added factor of hydrocephalus persisting, which was a feature of the present case. Some of these babies do, in fact, survive the initial hæmorrhage.

Report of a Case.

A male infant, of birth weight 8 lb., was born on October 12, 1958, at the Women's Hospital, Crown Street, Sydney, after a labour of 12 hours and an easy forceps delivery after the head had been showing for some time. The forceps were accurately applied by a senior obstetrician after the head had rotated well. For the first 24 hours the baby behaved normally. He then had a convulsive turn consisting of rigidity of the body, clenching of the hands and stiffening of the legs, without any localizing features. Later in the day the fontanelle was rather full, and further twitchings of the left hand were noted. The right eyelid drooped, and the lid could not be raised when the left eye was wide open. Mild left facial paresis was noted, and was thought to be possibly due to forceps pressure. The pupils were equal and reacted to light. Subdural taps were done. On the right side the result was indeterminate, as no fluid came from the needle, and on the left the result was negative, as clear fluid ran from the puncture when the needle was withdrawn.

On October 15 the child was listless and drowsy, vomiting all feeds, the fontanelle was tense and no fluid was obtained when the right subdural space was tapped. Lumbar puncture was attempted, but only 1 ml of thick blood came from the needle. This did not clot readily, and the supernatant fluid was xanthochromic.

On October 16 the fontanelle was more tense. Right subdural tap again gave an inconclusive result, and it was thought necessary to exclude a subdural hæmatoma over the right hemisphere. A burr hole was made under local anæsthesia, anterior to the ear in the line of the flap that might be necessary. When the dura was opened, a small amount of old blood was evacuated. Two other burr holes, one posteriorly and one superiorly, revealed no blood, and the brain was seen to be hard up against the dura. This excluded any large collection of blood over the right hemisphere. Through the fontanelle the right ventricle was punctured and found to be dilated, with a high cerebro-spinal fluid pressure. Twelve millilitres of clear fluid were removed, and 10 ml. from a similarly dilated left ventricle. After this, the fontanelle was soft and the baby's condition improved considerably. Lumbar puncture again showed only 1 ml. of thick, dark blood. Over the next four days the pressure was kept down by daily ventricular puncture, and on October 21 a needle ventriculogram showed moderate hydrocephalus; however, there was still a substantial amount of cortex anteriorly, the anterior horn being seven-eighths of an inch from the frontal bone. Lumbar puncture now showed brownish fluid blood, but only 2 ml. could be removed.

It seemed definite that there must be a large clot in the posterior fossa, producing obstructive hydrocephalus and spreading down the subdural space of the spinal theca, to account for the appearances at lumbar puncture. On October 25 posterior fossa craniotomy was carried out, and a large amount of old semi-solid blood was evacuated immediately the dura was opened. Recovery from the operation was satisfactory; but in spite of the relief given by decompressing the posterior fossa and evacuating the clot, the hydrocephalus was not relieved, and puncture of the fontanelle was necessary every day or so to relieve the pressure. Progressive lumbar punctures showed gradual clearing of the spinal fluid, and by October 30 the fluid ran freely, and it was realized that the hydrocephalus was now of the communicating type and would warrant a shunt operation if the brain had not been too severely damaged. Accordingly, air studies were carried out, which showed the anterior horn of the lateral ventricle five-eighths of an inch from the frontal bone, and this was thought to indicate a useful amount of brain substance.

On October 31, the operation of lumbar arachnoid supra-hepatic shunt was carried out, a polythene tube being brought from the spinal theca into the peritoneal cavity and anchored above the liver. This tube worked satisfactorily. The fontanelle remained depressed after operation, and the baby progressed satisfactorily. The head circumference at two months was 15·75 in., only slightly above the average for this age given by Dorothy Russell, and the fontanelle was flat.

Discussion.

The present report is that of a rarity, as it must be accepted that in the majority of cases in which hæmorrhage occurs in the posterior fossa, the condition is rapidly fatal. In those patients who survive the initial hæmorrhage, there are no focal signs to indicate that the posterior fossa is the site of hæmorrhage. In Coblenz's case (1940), as in the present case, the diagnosis was suspected by the occurrence of high intracranial pressure with a bulging fontanelle, together with internal hydrocephalus demonstrated by air studies. Hæmorrhage due to birth trauma should be distinguished clearly from the subdural hæmatoma of later infancy. There is little dispute that the former condition is due to head moulding; the actual site of the hæmorrhage will depend on which veins are ruptured. If a bridging vein over the parietal region is torn, there may be a localized hæmatoma in that region. If inferior veins are torn, the hæmatoma may be more extensive and may even spread over the whole of one hemisphere. This is probably not a massive single hæmorrhage, but a slow spread from a bleeding point. Bilateral hæmorrhages occur, and one must assume that in these cases the stresses of head moulding have affected both sides; but these cases are not so common. When a large vein—for example, the great cerebral vein—is torn, or when a tentorial tear extends into a venous sinus, the hæmorrhage is catastrophic and rapidly fatal. Thus the same mechanism is responsible for a variety of clinical pictures, ranging from a small parietal hæmatoma which may absorb without leaving any sequelæ to a posterior fossa hæmorrhage with fatal consequences. The present case illustrates the frank impossibility of predicting from the history or even from the nature of the labour what type of labour will produce "birth injury", and shows how valueless is a retrospective history of a difficult labour in estimating the place of birth trauma in the causation of hæmatomas diagnosed at a later date. This child was apparently normal at birth, and the labour was not classed as "difficult", there was no difficulty in resuscitation but the symptoms appeared in 24 hours. There must be many babies born after a "difficult" labour who do not develop subdural hæmatomas.

The cause of the subdural hæmatomas of later infancy is not so clear. Virchow's original concept of pachymeningitis hæmorrhagica interna has been abandoned, and, if those cases associated with major trauma in which the causation is clear are disregarded, the usual explanation offered is that trauma has occurred which may be so slight as not to be remembered. That such a mild degree of trauma can tear a vein entering the longitudinal sinus seems most unlikely, and we must look for another explanation of many of these cases.

This group is characterized by a more constant clinical pattern than the neonatal cases, but it is quite possible that there are a number of different causes. In these cases the hæmorrhage is usually bilateral. The constant pathological finding is the presence of false membranes between the dura and arachnoid. These are not formed by an inflammatory process, as Virchow originally postulated, but, according to Inglis (1946), represent a reaction on the part of modified connective tissue cells lining the dura mater to the presence of extravasated blood in the subdural space. Of the nine cases (all in adults) described by Inglis in which membranes were found, only one was definitely associated with trauma. The causes of the others varied from leukæmia to an obscure illness associated with optic atrophy; but the possibility that trauma was a contributory cause in some of these could not be altogether excluded. The present thinking on the causation of subdural bleeding in infants is dominated by the idea of trauma. But there are other causes of hæmorrhage, and Ingalls (1936) drew attention to the association of scurvy with some cases of post-natal subdural hæmatoma, a term which he defined as being synonymous with the pachymeningitis hæmorrhagica interna of the older writers. He suggested that scurvy might be the underlying basis or "bleeding diathesis" that enabled mild trauma to cause rupture of a cortical vein. But why is it necessary to consider trauma at all when there is no history of trauma? In disregarding trauma as a necessary factor, Baar (1957) has taken us full circle back to the concept of pachymeningitis hæmorrhagica interna in infants, due to degenerative, vasoproliferative or inflammatory changes in the dura. The clinical picture is that of an underweight infant, aged four to eight months, developing slow enlargement of the head with vomiting, crying and somnolence and the occurrence of retinal hæmorrhages. These infants have membranes over the surface of the brain and xanthochromic fluid between its layers. These cases present, in fact, the same picture as many of the cases reported by Ingraham (1954) in his series of subdural hæmatomas, and are not due to birth injury or post-natal trauma. Our experience in Sydney has been that we have encountered many more cases of subdural bleeding in the first month—and undoubtedly due to birth trauma—than we have in the later months of infancy, whatever the cause of these may be. Many of these later cases could be due to a degenerative disease of the dura, and it is not necessary to conclude that every infant who has this condition has sustained a head injury. Whatever our feelings about causation, the surgical problem remains the same, and where blood or membranes containing fluid are found in the subdural space, they should be removed.

The question is not entirely an academic one, as at present a number of these cases are attributed to the sequelæ of birth trauma without sufficient evidence. It is also evident that a complacent attitude in explaining all these cases on the basis of trauma will prevent the research that is necessary to explain this controversial problem fully.

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PURE PULMONARY VALVE STENOSIS.[1]

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THE intention of this paper is to present a congenital cardiac lesion of increasing importance, one of relatively ready clinical diagnosis, and one which lends itself to surgical correction to a highly satisfactory degree.

The term "pure pulmonary valve stenosis" implies an obstruction, variable in degree, at the pulmonary valve site due to a misshapen valve of curiously uniform appearance. The term strictly debars any suggestion of an added ventricular septal defect—i.e., a Fallot-type lesion. A concomitant auricular septal defect, unless gross, has less significance, for with valvotomy its effect may be minimal.

In pure pulmonary valve stenosis, the basal ring is narrowed, the cusps are ill-formed, adherent and thickened; the valve presents as a truncated cone with a small apical or eccentric orifice, the diameter of which determines the degree of obstruction and therefore the symptoms. The consistency of the valve tissue varies; there is evidence that with the passage of time from infancy onwards increased thickening and rigidity of the valve develop, which in turn probably increase the obstruction. Gross has suggested failure of the embryonic vessels supplying the valve cusps as a cause of the thickening and lack of cusp differentiation.

The right ventricle is always hypertrophied, and in the final stages dilated, as indeed are all chambers. With increasing dilatation the tricuspid valve becomes incompetent, and this, with the added difficulty of complete systolic emptying of the right ventricle, will force a shunt from the right atrium through the foramen ovale or a preexisting atrial septal defect into the left atrium. A right-to-left shunt with consequent cyanosis will now appear in what was originally a non-cyanotic lesion; exercise may produce this effect temporarily. The veins in the neck reflect the extension of right ventricular embarrassment, and may show giant a-waves. Towards the end, the liver will enlarge and may pulsate, a clinical finding difficult to demonstrate convincingly. The left ventricle, its input diminished, remains relatively small.

Tricuspid incompetence was strikingly demonstrated at operation in two of our cases by the occurrence over the right atrium of a marked systolic thrill, which vanished immediately after valvotomy.

Post-Stenotic Dilatation.

Immediately distal to the stenotic valve there occurs the phenomenon of post-stenotic dilatation. This is of interest and great clinical importance. First observed distal to the constriction produced in the subclavian artery by a cervical rib and a tight scalenus anticus muscle, it is now a familiar sight in angiocardiography, and in surgery upon such lesions as pure pulmonary stenosis, aortic stenosis and coarctation of the aorta. Holman (1954) has described what is undoubtedly the correct explanation. When a mass of fluid at a given velocity passes through a constricted area, its velocity is increased at the site of constriction by a degree inversely proportional to the square of the diameters of the normal and the constricted channels. This high-velocity jet emerging from the stenotic site strikes the low-pressure slowly-moving blood mass and immediately sets up a series of eddy currents, some of which actually travel in reverse direction to the axial flow. There is an abrupt transference of the axial to a lateral force and of kinetic energy into potential energy. The continuation of these hydraulic forces over a long period, and particularly their intermittent nature, are responsible for structural fatigue in the wall of the vessel at the site of the eddying currents. Dilatation occurs as an inevitable result. The effect has been produced experimentally with rubber tubing.

The phenomenon has great clinical importance, especially in regard to diagnosis in pulmonary stenosis. The pulmonary artery in the tetrad of Fallot never shows post-stenotic dilatation of any degree, because the septal defect allows a deviation of the pulmonary stream into the aorta. Clinically, the dilatation is often palpable as a systolic lift in the second left intercostal space, and as the jet bursts into the dead space with each systolic contraction of the hypertrophied right ventricle, a loud "ejection" slap, sometimes called the spinnaker flap, initiates the first sound and is followed by the characteristic bruit. The surprising force of the lateral thrust at the dilated area is very evident at operation and under the X-ray screen, and is responsible for the wide distribution of the thrill—so wide at times that it carries to the aortic area and great vessels of the neck, and may simulate an aortic stenosis.

This dilated area is of convenience to the surgeon, in that it allows "slack" for the various manœuvres of the operative procedure.

It is of interest that there appears to be no fixed relation between the degree of post-stenotic dilatation and the severity of the stenosis. In many apparently mild cases considerable post-stenotic dilatation is seen radiologically. We have much to learn; such cases may not be as mild as they appear.

Further, it has been noted by Dr. H. G. Hiller that on straight films, and in fluoroscopic screening and angiocardiographic examination of three of our patients with high right ventricular pressures (150 to 170 mm. of mercury), post-stenotic dilatation was apparently not present as a definite localized phenomenon. We do not yet know the explanation of this variation from the routine findings.

A feature which can cause confusion in catheter studies, especially after operation, is the hypertrophy of two bands of muscle in the infundibulum, the crista supraventricularis and an extension of the moderator band. The result is to produce an infundibular obstruction which is a secondary effect of the basic stenotic lesion. After operation, with a collapsed ventricle, this acquired infundibular stenosis may produce disconcerting pressure tracings.

The actual output of hearts with pure pulmonary valve stenosis is of interest. Many patients at rest show a normal output in spite of the stenosis. Exertion, however, highlights the inability of the right ventricle to overcome the rigid valve obstruction, and the output will not rise normally. Lurie and Shumacker (1953) have demonstrated an interesting comparison of an apparently symptomless patient with a normal output at rest rising very little with exercise, and a severely affected patient with grossly limited output at rest rising well with exertion—after valvotomy.

The Symptoms.

There are only three symptoms in this condition, limitation of effort, dyspnœa and, in the severe cases in which there is a forced intracardiac shunt, central cyanosis. Peripheral cyanosis due to diminished cardiac output may occur, and as an added factor to the central cyanosis it can produce a startling degree of cyanosis (Figure I).

As may be expected, there is great variation in the severity of the symptoms. The size of the aperture in the deformed valve will be the prominent factor in their production. An infant may gasp out its life in a few days;

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

other patients may carry on to adult life with relative ease.

Squatting, which is such a feature of the tetrad of Fallot, is rarely seen in pure pulmonary stenosis, and its absence actually serves as a useful diagnostic aid.

All the patients in our series with severe symptoms had high pressure gradients from right ventricle to pulmonary artery; but several patients with high pressure gradients proclaim no symptoms. Time, of course, is a factor. It is correct to assume that a child of 14 years, however symptomless she may appear, who has a pressure gradient across her stenotic pulmonary valve of 125 to 17 mm. of mercury, will assuredly develop symptoms within a few years. We have had this experience.

The Signs.

The lesion is essentially non-cyanotic. A reversed intracardiac shunt through a forced foramen ovale or an actual auricular septal defect will produce persistent central cyanosis. Exercise may produce it temporarily. Peripheral cyanosis may occur because of diminished flow.

Clubbing of the digits, polycythæmia and high hæmoglobin values will develop if central cyanosis is persistent.

The precordium is prominent in severe cases owing to the right ventricular hypertrophy, and a right ventricular heave is palpable. At the pulmonary area there is a thrill, and the impulse of the post-stenotic dilatation may be palpable. Transmission of this impulse may be wide, and may extend to the jugular fossa and carotid trunks, thus simulating an aortic lesion.

Auscultation reveals a long, harsh, systolic bruit at the pulmonary area, usually initiated by a loud "ejection slap". This bruit, which is one of the most constant and helpful diagnostic features, tends to be conducted upwards rather than downward—a point of distinction from the murmur of the ventricular septal defect. The second sound is usually single, soft and muffled or practically inaudible; but it is possible, in mild cases, for the pulmonary second sound to appear normally split.

Blood pressures are non-specific, and tend toward low systolic and low-pulse pressure recordings.

The electrocardiogram shows a right ventricular hypertrophy pattern in practically all cases. Evidence of increasing strain on the right side of the heart may be demonstrated by an increasing right ventricular hypertrophy pattern and *S–T* segment depression in lead III, and may be helpful in assessing the need for operation. The *P* wave in lead II has been high and peaked in all the cyanotic patients.

In mild cases, radiological examination may reveal no abnormality; but it is striking how frequently post-stenotic dilatation is seen even in such cases (Figures V and VI).

The classical picture, seen better with fluoroscopy, is that of a generally enlarged heart, a large right auricle, a large ventricular mass not tip-tilted, and a very prominent and forcibly pulsating main pulmonary artery (at the site of the post-stenotic dilatation), with strikingly "quiet" main branches (Figure VII). In one of our severe cases this post-stenotic dilatation was not obvious on the X-ray plate, but was very clear at operation. The lung fields are notably oligæmic. The aortic arch has been left-sided in all our cases. As cardiac embarrassment increases, the heart tends to become globular, and radiologically the post-stenotic dilatation may be submerged in the cardiac mass (Figure VIII). Pure pulmonary stenosis has no safety valve except that of forcing the tricuspid valve, and this occurs only after cardiac dilatation and radiologically evident enlargement of the heart have occurred. All cyanotic patients therefore show cardiac enlargement, which is unlike the tetrad of Fallot, and this serves as a useful differentiating feature.

Differential Diagnosis.

Whilst there are several conditions which clinically bear a superficial resemblance to pulmonary valve stenosis, there is only one which provides practical difficulty.

This is the case in which a ventricular septal defect is also present—i.e., a Fallot type of pulmonary stenosis exists. Such patients can be virtually non-cyanotic, which adds to the difficulties.

Since the surgical attack in the tetrad of Fallot is a formidable intracardiac radical procedure under cardiac by-pass, whilst that of pure valve stenosis is a supravalvular transarterial approach under hypothermia, it can be understood that differentiation of the two conditions is vital. Valvotomy in the somewhat rare tetrad of Fallot in which a valve stenosis is present is only half the problem.

The features which help differentiation are as follows.

1. Persistent central cyanosis is much more the hallmark of the tetrad of Fallot; but with forced interatrial shunt, it may be present in pure valve stenosis. The patient with the tetrad of Fallot may, however, be virtually non-cyanotic.

2. Squatting, which is such a feature of the tetrad of Fallot, is rare in pure valve stenosis.

3. The clinical findings—e.g., right ventricular impulse, pulmonary systolic bruit, faint or absent pulmonary second sound, right ventricular hypertrophy pattern in the electrocardiogram—can be identical, but a definite ejection slap would favour valve stenosis.

4. The radiological picture is often decisive, for the following reasons. (*a*) The heart in Fallot's tetralogy tends to be small, because of its safety valve, the septal defect. (*b*) A right-sided aorta—i.e., arching to the right of the trachea—is common in Fallot's tetralogy; it is practically never seen in pure valve stenosis. (*c*) Post-stenotic dilatation is absent in Fallot's tetralogy, but, with the reservation mentioned earlier, is the hallmark of pure valve stenosis. One of our patients with tetrad of Fallot had a valve stenosis only, which is rare, and a slight degree of post-stenotic dilatation exists in this case.

The cardiac catheter is highly discriminating, and will demonstrate an abrupt pressure gradient at the valve site. The fact that right ventricular pressure approximates systemic pressure is not always decisive, for this may occur in pure valve stenosis. A right ventricular pressure above systemic pressure is more in favour of pure stenosis than of tetrad of Fallot. Infundibular stenosis greatly favours a diagnosis of tetrad of Fallot.

The selective angiogram, in which opaque medium is forced directly into the right ventricle, is a valuable diagnostic weapon, and can demonstrate the following clearly: (*a*) the cone-shaped valves of pure stenosis (Figure X); (*b*) the actual jet passing through the valve (Figure X); (*c*) the post-stenotic dilatation of pure stenosis (Figure X); (*d*) the aorta filling simultaneously with the pulmonary artery in tetrad of Fallot, without the dye passing first from right atrium to left atrium which can occur in pure valve stenosis (Figures XI and XII); (*e*) a simple puff of dye from right ventricle to left ventricle, which demonstrates that a ventricular septal defect is present (Figure XIII).

Other lesions which may cause confusion, especially when they are mild, and especially when they occur in infants, are the following:

Ventricular Septal Defect.

In ventricular septal defect the bruit is similar, but it is lower and is not transmitted cranially. There may be some ejection sound. The pulmonary second sound is usually split and loud. Pulmonary plethora is present.

Atrial Septal Defect.

This lesion should rarely cause confusion, as the bruit is much softer and the second sound louder, and pulmonary plethora is present.

The Patent Ductus.

One of our patients was actually operated on elsewhere with the diagnosis of a patient ductus; the post-stenotic dilatation, no doubt because of its forcible pulsation, suggested a left-to-right shunt. The bruit, however, was purely systolic. In infants, however, confusion can arise.

Aortic Stenosis.

Because of the wide propagation of the murmur and thrill of pulmonary valve stenosis, confusion can occur with aortic stenosis. The pulse pressure can be low in both lesions. The electrocardiogram is normally left-sided in these cases. In all these cases the catheter examination is usually decisive.

The Series.

There have been 61 patients (37 girls and 24 boys) with pure pulmonary valve stenosis in our series since 1954. They divide into two almost equal groups, as follows. (i) Those with no symptoms and right ventricular pressures of approximately 50 to 60 mm. of mercury or under; there were 32 of these patients. (ii) Twenty-nine patients with pressures above 70 mm. of mercury. Nine of these showed gross limitation of effort with cyanosis due to a forced foramen ovale or actual atrial septal defect shunt. One of these died undiagnosed as an infant, one died at operation by a transventricular approach without hypothermia, and one died after operation with jaundice and liver failure of uncertain ætiology.

In the 20 cases remaining, symptoms were variable and usually quite mild.

Whilst all the patients with symptoms had high pressures, the reverse was not always true; but the time factor was strikingly demonstrated by two patients, who spontaneously reported decreasing exercise tolerance over a period of three to four years.

Seventeen patients have been operated upon by the transarterial approach under hypothermia by Mr. Russell Howard, Mr. Peter Jones and Mr. N. Myers, and one girl of 16 years by Mr. Ken Morris at the Alfred Hospital under "Fluothane" anæsthesia.

With regard to the results of operation, it is not always easy to assess that somewhat nebulous expression "great improvement", especially when symptoms may not have been very severe. Certain psychological factors, and an anxious desire of the parents to see improvement at all costs—even of veracity—can make assessment difficult.

It is what can happen in the severe cases that is so convincing, and that renders it reasonably safe to assume that this operation is one of great value and is certainly life-saving at times.

In the two cases in which post-operative catheter studies were made, figures in Table I indicate the change in right ventricular pressure.

TABLE I.

Pressure (Millimetres of Mercury).	Case I.	Case II.
Before operation:		
Right ventricle	195	170
Pulmonary artery	30	20
After operation:		
Right ventricle	46	55
Pulmonary artery	10	25

One female child, aged 20 months, grossly cyanotic, with clubbed digits and disabled with a large heart, had an oxygen saturation by the ear oximeter of 84%. The catheter would not enter her pulmonary artery because of the small valve orifice. Her right ventricular pressure was 100 mm. of mercury. After operation the oxygen saturation lifted to 96% and her colour is normal. The parents' statement that she is "wonderfully improved" can be believed (Figures I, II, III, IV).

An older boy, aged eight years, grossly disabled and becoming increasingly cyanosed, had the largest heart we have seen in the series, with a gradient of 135 mm. of mercury in the right ventricle to 5 mm. of mercury in the pulmonary artery. He has shown a most dramatic decrease in the size of his heart (Figures VIII and IX). From a timid lad who always walked at a snail's pace and was always carried up and down stairs, he has changed into one capable of playing football. His post-operative catheter study failed.

Three patients show pulmonary diastolic bruits since operation. In view of the fact that dogs survive indefinitely after complete ablation of the pulmonary valve, it would seem that there is little to worry about in this regard. It is unlikely also that the valve incisions will fuse; the constant dilating effect of the jet through the valve should prevent this.

What should be the criteria for operation, now that the procedure has become firmly established? There is no doubt about group (ii), with high right ventricular pressures with or without symptoms. It seems certain that decompensation will occur, and it has been clearly observed in two cases at least.

Group (i) presents greater difficulty in making this decision. One child, quite symptomless, has a heart of normal size, a right ventricular pressure of 40 mm. of mercury, a balanced electrocardiogram, but very well-marked post-stenotic dilatation (Figure V). It is not possible to say what will happen to this heart; but it may well prove to be correct to operate on the valve whilst the child is young. Increased rigidity of the valve with the passage of years may produce symptoms and signs not as readily relieved as in childhood. We do not yet know accurately the long-term future of this group.

Summary.

1. The pathological anatomy and physiology and certain hæmodynamic effects of pure pulmonary valve stenosis are described.

2. The symptoms, signs and differential diagnosis, especially from the tetrad of Fallot, are indicated.

3. A group of 61 cases, in which varying clinical responses to the lesion were shown, is presented, and an attempt is made to show objectively that the effect of valvotomy is beneficial.

4. The decision in regard to surgery in the symptomless low right ventricular pressure group is still difficult, and can be clarified only with further experience.

Acknowledgements.

Dr. Harry Hiller and Dr. Alex Venables have done all the catheter and angiogram work in these cases. Without their careful and critical work, the presentation of this series and its documentation would not have been possible. I am grateful to the various members of the senior medical staff of the Royal Children's Hospital, who have referred their patients for study. Mr. C. Murphy and Miss E. C. Omond, of the Photographic Department of the Royal Children's Hospital, have been tireless in their efforts to reproduce the X-ray pictures and photographs accurately for projection and publication. The Kodak Company has been most helpful in producing colour prints from "Kodachrome" transparencies.

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INHALATION OF EXCESS LIQUOR AS A FACTOR IN THE PRODUCTION OF HYALINE MEMBRANE ATELECTASIS.[1]

By KATE CAMPBELL,
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To the clinician, the primary problem in hyaline-membrane atelectasis is its prevention, and this necessitates a knowledge of its causation. To the investigations on ætiology being carried out by laboratory workers can be added the bedside observations of the pædiatrician in the neonatal period.

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

CLINICAL OBSERVATIONS.

The following five points impress the clinical worker: (i) the frequent association of hyaline membrane atelectasis with conditions in which the fœtus or infant inhales more liquor than normal; (ii) its high incidence in prematurity; (iii) the characteristic type of respiration; (iv) the presence of œdema and sudden attacks of cyanosis; (v) the self-limited course of the condition. I will consider these five points briefly, first from the clinical and then from the theoretical aspect.

Inhalation of Excess Liquor.

Conditions under which more liquor than normal is aspirated may occur under the following circumstances.

Before birth, when the fœtus is exposed to oxygen lack while the membranes are intact, the strong respiratory efforts produced in utero cause the inhalation of much liquor—e.g., accidental hæmorrhage, placenta prævia or any condition causing fœtal distress.

At birth, when there is more than the normal amount of liquor present at the time of the first breath (as in Cæsarean section, particularly elective Cæsarean section), there is no "chest squeeze" by the birthpassage to rid the airways of liquor, and in vertex presentation the infant is extracted with the head up instead of down.

Excess liquor is inhaled also in hydramnios, and in cases in which the membranes rupture just at or immediately before birth—as in second twins and second and third triplets. In these cases, frequently a large gush of liquor follows the expulsion of the infant, and his first breath may be taken in a veritable waterfall.

High Incidence in Prematurity.

The condition is rare in infants weighing under 750 grammes (1 lb. 10 oz.) at birth, and its maximum incidence is in the group weighing 1250 to 1500 grammes (2 lb. 12 oz. to 3 lb. 5 oz.). This is therefore a group with a specific character. The incidence decreases sharply from 2000 grammes (4 lb. 6·5 oz.).

Characteristic Respiration.

Infants with hyaline membrane atelectasis have usually the typical "grunting" type of respiration, and on inspection present a typical picture. The chest is full and remains in the fixed inspiratory position—rather like the chest of an asthmatic—with extremely little tidal movement. Inspiration is short. At first there is retraction of the xiphisternum with inspiration, so that in silhouette the chest looks like that of a pouter pigeon. Later, the lower sternal area shows marked inspiratory crateriform retraction, with mild to minimal lateral retraction. Expiration is prolonged and moaning, and appears difficult, terminating in a forcible grunt.

As the lungs contain less air than normal, it would appear that the fixed inspiratory position is due to engorgement with blood, and that it is this which diminishes the pulmonary compliance. This suggests the presence of a circulatory factor.

Œdema and Cyanosis.

Generalized œdema is constantly present. Cyanosis from the pulmonary condition is to be expected, but the sudden attacks of cyanosis are interesting. Some of these are undoubtedly due to obstruction with mucus, but in others no such obstruction is found.

Also in the course of the disease quite organic-sounding bruits may develop, which disappear when the infant's condition improves. These observations suggest a circulatory factor.

The Self-Limited Course.

The self-limited course is striking. When death occurs from hyaline membrane atelectasis, it is almost always before 48 hours. Occasionally, with energetic treatment, in fatal cases death may be postponed till the fourth day. Usually improvement in survivors begins at 72 hours. This suggests a mechanism for removal of the membrane.

Suggestions from Clinical Observations.

The clinical observations therefore suggest the following as factors in ætiology: (i) the presence of more liquor in the lung than can be removed; (ii) œdema and circulatory disturbance producing œdema fluid in the lung. One therefore hypothesizes the interaction of the thromboplastic effect of the unabsorbed liquor on the œdema fluid, with the formation of fibrin, as the basis of membrane formation. There is a strong body of research opinion which is of this belief.

POSSIBLE MECHANISMS OF PRODUCTION OF HYALINE MEMBRANE ATELECTASIS.

Excess Liquor Remaining in the Lungs.

There must normally be some liquor in the respiratory passages, and this is removed by vascular absorption. The absorption will depend (*a*) on the amount of liquor present, (*b*) on the development of the pulmonary vascular bed and (*c*) the rate of pulmonary blood flow.

It has been shown (Dawes, 1958) that the rate of pulmonary blood flow depends on the degree of ventilation of the lung with air. Therefore the infant with poor initial lung expansion and consequent impaired blood flow will be less able to remove the liquor. Even a normal amount of liquor in the lung under such conditions could become abnormal.

Thus, either excess aspiration or impaired removal of liquor or both could cause unabsorbed liquor to remain in the alveolar ducts and alveoli.

Presence of Œdema Fluid in the Lungs.

The œdema fluid with which the liquor interacts could be produced by the following different mechanisms.

1. By the pulmonary hypertension of the transitional circulation of the new-born. It is accepted that for a variable time after birth the ductus remains functional permitting a flow from aorta to pulmonary artery. This extra blood reaching the lung could tend to produce pulmonary hypertension.

2. By a plasma shift. Gairdner *et alii* (1958) estimate that a plasma shift of about 16% from the vascular compartment occurs very soon after birth, and in this the lung shares.

3. By hypoxæmia producing œdema.

TABLE I.

Subjects.	Weights.							Total.
	750 to 999 Grammes (1 lb. 10·5 oz. to 2 lb. 2·5 oz.)	1000 to 1249 Grammes (2 lb. 8 oz. to 2 lb. 11·5 oz.)	1250 to 1499 Grammes (2 lb. 12 oz. to 3 lb. 4·5 oz.)	1500 to 1749 Grammes (3 lb. 5 oz. to 3 lb. 13·5 oz.)	1750 to 1999 Grammes (3 lb. 14 oz. to 4 lb. 5·5 oz.)	2000 to 2499 Grammes (4 lb. 6 oz. to 5 lb. 7·5 oz.)	2500 Grammes and Over (5 lb. 8 oz. and Over.)	
Hyaline membrane atelectasis subjects ..	19	24	28	24	16	11	13	135
Controls	63	33	24	17	14	24	48	223

Removal of the Membrane.

Removal of the membrane is thought to be due to the fibrinolysin enzyme system and the activity of the leucocytes. Phillips *et alii* (1958) report that the fibrinolysin enzyme system of cord blood is low as compared with the normal, and that it tends to decrease with decreasing fœtal age. Thus premature infants would have an impaired mechanism for the removal of the membrane.

Factors in Prematurity.

In the premature infant, several factors are present which could predispose to hyaline membrane atelectasis. They are as follows: (i) Absent cough reflex, permitting aspiration of excess liquor. (ii) Poor pulmonary ventilation with diminished pulmonary blood flow, leading to impaired removal of liquor. (iii) The larger placenta in proportion to the fœtus in the premature than in the full-time infant. If the cord is not tied till after pulsation ceases, the infant will get relatively more blood from the placenta, and so the possibility of circulatory embarrassment and pulmonary œdema exists. (iv) Diminished fibrinolysin leading to impaired removal of any membrane formed. It is suggested that the summation of these factors may have some influence in bringing about the increased incidence of hyaline membrane atelectasis in prematurity.

Clinical Application.

It seems to me, therefore, that while several factors play a part in the ætiology of hyaline membrane atelectasis, the presence of unabsorbed liquor in the lungs is a necessary factor for the development of the membrane. If we could prevent this, we could remove one necessary link in the chain of events and so prevent the condition from occurring.

We cannot influence the rate of absorption of liquor from the lungs, but we can adopt practical measures to minimize the amount of liquor inhaled at birth.

Present Investigation.

In order to test the validity of the theory that the inhalation of excess liquor was a factor in the production of hyaline membrane atelectasis, I examined hospital statistics to see if hyaline membrane atelectasis did occur more frequently in cases in which this possibility existed.

I took accidental hæmorrhage and placenta prævia as two conditions in which the fœtus is subjected to oxygen lack with membranes which are practically always intact, and Cæsarean section as a condition in which there is usually more liquor present at birth than normal. These were the only three conditions in which I thought the necessary information would be accurately recorded in histories. They represent only a portion of the cases in which inhalation of excess liquor could occur.

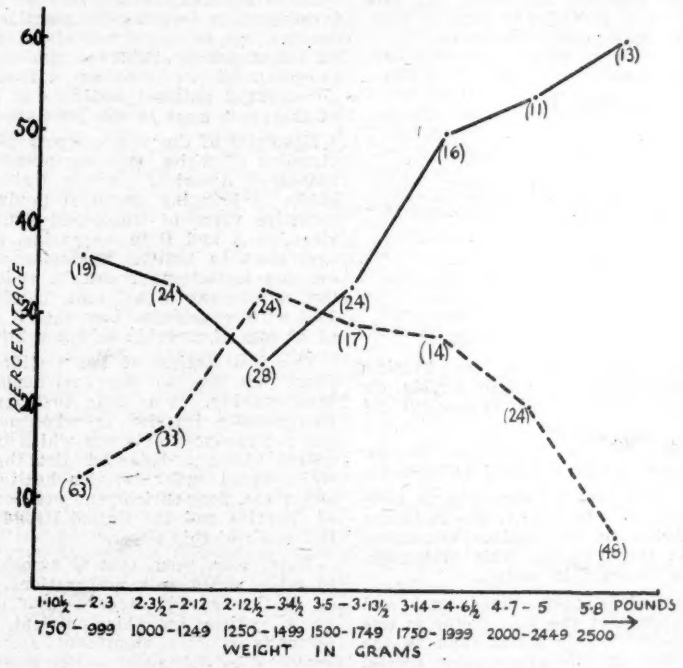

FIGURE I.

Solid line, percentage of cases of hyaline membrane in which the excess liquor factor was present. Interrupted line, percentage of controls showing excess liquor factor. Figures in parentheses, number of infants in each weight group.

Material.

The material was taken from the records of the Royal Women's Hospital, Melbourne, for the four years 1955 to 1958 inclusive. All those cases were included in which it was stated that some of the alveoli contained hyaline material, though hyaline membrane atelectasis was not necessarily the cause of death in all cases.

In all cases the diagnosis was confirmed by post-mortem histological examination, and in practically every case by one pathologist, Dr. R. Barter. It was found that under birth weight 750 grammes (1 lb. 10·5 oz.) hyaline membrane atelectasis was a rarity, the lungs being pale pink and airless. Therefore infants under 750 grammes' birth weight were not included.

The earliest age at death was one and a half hours, and all infants had died before 48 hours.

On the advice of the Commonwealth Statistics Department, I have taken as controls all post-mortem records of live-born infants weighing 750 grammes and upwards born over these four years who lived from one and a half hours to just under 48 hours. I have called the three conditions of accidental hæmorrhage, placenta prævia and Cæsarean section the excess liquor (E.L.) factor, and estimated in what percentage of cases it occurred in each weight group in both the hyaline membrane atelectasis series and the control series.

The cases were divided into groups of 250 grammes' birth weight up to 2000 grammes, then 2000 to 2499 grammes, and the last group was 2500 grammes and over. This gave seven groups.

It is recognized that the numbers are small, and this is therefore to be taken as a preliminary report. Suggestions based on it are consequently tentative.

Results.

The results are set out in Table II.

TABLE II.

Subjects.	Total Number.	Number Showing E.L. Factor.
Hyaline membrane, atelectasis series	135	52 (38·5%)
Controls	223	39 (17%)

The incidence of the E.L. factor in the hyaline membrane atelectasis series was more than double its incidence in the controls. This would appear to be significant.

On breaking up the figures into 250-gramme groups, the following unexpected graph was obtained (Figure I).

It is seen that in all groups except the 1250 to 1500-gramme group (2 lb. 12 oz. to 3 lb. 5 oz.), the incidence of the E.L. factor is higher in the hyaline membrane atelectasis series than in the controls. This preponderance increases with each increase in weight.

In the 1250 to 1500-gramme group—the largest and most vulnerable—the incidence of the E.L. factor is less in the hyaline membrane atelectasis series than in the controls. This suggests that in this group some special factor is operating. It is at this period of gestation (30 to 32 weeks) that there is clinically the greatest amount of liquor present relative to the size of the fœtus. (It is for this reason that this period is chosen for the diagnostic amniotic tap.) Under such circumstances there would be always a larger amount of liquor present at the birth than usual so that the E.L. factor would be present in all infants of this group.

I would suggest that this is why the highest incidence of hyaline membrane atelectasis occurs in this group, and accounts for the discordance between this and the other five groups.

CONCLUSION.

In conclusion, it would seem that this series allows one tentatively to suggest the possibility that inhalation of excess liquor may be a factor in the production of hyaline membrane atelectasis, and that this factor increases in importance with increasing fœtal age.

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SHOULD ASCORBIC ACID BE ADDED TO INFANT DRIED MILK PREPARATIONS?[1]

By F. W. CLEMENTS,
Sydney.

IT was suggested by a number of our members that a discussion on the subject of the addition of ascorbic acid to dried milk infant foods would be topical in view of the recent mild epidemic of scurvy in Victoria, and of the suggestion of a group of pædiatricians that this measure should be considered as a means of preventing the development of scurvy in young infants.

There are two justifications for the addition of a nutrient or chemical compound to a foodstuff: (i) to replace the nutrient or nutrients lost in the commercial processing, or to bring the nutritive value of a particular product up to the level of an alternative one which is either not available or too expensive for a section of the people; (ii) to use a foodstuff as a vehicle for an essential nutrient which is in low supply in the diet of the great mass of the population.

Examples of the first category are the enrichment with vitamins of white flour for bread-making in the United States of America and the United Kingdom, with the object of bringing the final product up to at least the nutritive value of wholemeal flour and the addition of vitamins A and D to margarine, making it nutritionally equivalent to butter. Examples of the second category are the iodization of salt in endemic goitre areas, and the enrichment of all milk powder in Greenland since 1956 with ascorbic acid, so that it contains the equivalent of 50 mg. of ascorbic acid per half litre (Uhl, 1957).

The justification of the fortification of margarine is clear, but not so the enrichment of white flour for bread-making. It is true that on paper white flour is nutritionally inferior to wholemeal flour; but no one has yet produced evidence which conclusively proves that people eating a balanced diet that includes unenriched white bread suffer inferior health. Much could be said about the flour-enrichment policies of the United States of America and the United Kingdom, but this is outside the scope of this paper.

It is clear, then, that if ascorbic acid is to be added to infant dried milk preparations, the justification would be in the second category—that is, milk would be used as a vehicle for this nutrient, since milk does not normally contain significant amounts of ascorbic acid. Justification for such action must then be in terms of the community needs and not in respect of the foodstuff.

Greenland is in a unique position, in that the supplies of foodstuffs rich in ascorbic acid are limited in quantity, variety and season of the year for which they are available. Conditions there are in no way comparable with conditions in Australia, where there is throughout most of the year a plentiful supply of foodstuffs rich in ascorbic acid.

Breast milk contains up to 5 mg. per 100 ml. of ascorbic acid, so that the infant of six months of age who is wholly breast fed will obtain about 35 mg. of ascorbic acid, although the diet of the mother has a marked influence on the level of ascorbic acid in her milk. Scurvy is extremely rare in the breast-fed infant, all cases described in recent literature having occurred in infants who have been artificially fed for some months.

The usual advice offered in Australia to the mother of both breast-fed and artificially-fed infants is that a supplement of orange juice should be given, starting with small amounts and working up rapidly, especially for artificially-fed infants, to two ounces per day. It is also the practice of some authorities to advise mothers to give a proprietary vitamin mixture containing ascorbic acid. In Australia the recommended daily allowance

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

of ascorbic acid for infants is 25 mg. Other fruits and fruit juices, such as tomato juice, black currant juice and pawpaw, are also used as sources of ascorbic acid, but the choice for the majority of mothers rests on orange juice.

The supply of oranges is not constant throughout the year, there being a trough in the late summer and autumn, when some oranges tend to be bitter, dry and generally a "bad buy" as a source of juice for infants. We have not yet developed the practice of using canned orange juice, a product widely and extensively in use in the U.S.A. This has the advantage of being uniform in consistency, taste and vitamin value. In England bottled "national" orange juice of specified vitamin value is distributed through the welfare centres.

It is widely recognized that some infants reject orange juice during the off season, undoubtedly because of its taste. This, however, is not a universal experience, and we can only conclude that the successful mothers have a better technique of preparation and presentation; perhaps the secret is the judicious use of sugar and dilution as well as the progressive introduction of a less palatable form—in other words, a higher standard of mothercraft. When the infant completely rejects orange juice, the majority of mothers know they can and should substitute a multivitamin preparation or ascorbic acid in tablet from—a further example of the possession of the correct information and its use at the appropriate time.

In her study of a number of infants with scurvy in Victoria, Dr. Jan Finney (1958) reported that 34 out of 48 infants investigated had not received any orange juice. The reasons for not giving the orange juice were as follows: 16 babies refused juice when it was offered, and the mothers had not persisted; 11 mothers did not buy oranges because of the cost; six mothers were New Australians, and to give orange juice was not the custom in their country; one mother was an epileptic and unable to care properly for her children. Dr. Finney noted that some mothers were unaware of the necessity to give ascorbic acid to an artificially-fed infant; they stated that they had successfully breast fed an older child, and felt confident that they could manage this particular baby without advice from a baby health centre. Fourteen infants had never attended an infant welfare clinic; the mothers of nine scorbutic infants had attended one irregularly. For those who had visited the centre, we would be justified in concluding that the health education had been faulty or inadequate.

Woodruff (1956) reported a similar set of circumstances in his inquiry into the causes of scurvy in Nashville, Tennessee. Deas (1956), in a review of the uptake of welfare foods in Edinburgh in 1955, found that 87·5% of infants three months of age and 92·6% of infants nine months of age were receiving some vitamin C supplement. Of these, less than half were receiving a daily dose equal to or greater than the recommended 25 mg. The same story has recently been reported from Canada (Grewer, 1958; Whelan *et alii*, 1958) and from Germany (Ocklitz, 1956).

Thus it will be seen that the problem of ensuring that all artificially-fed infants receive an adequate dose of ascorbic acid is widespread, and it seems ever-present. It is, however, not an extensive problem, in that the number of infants who are not given an ascorbic acid supplement as judged by the incidence of scurvy must be small. It can be considered that failure to give the requisite vitamin supplements is an expression of defective mothercraft, and must be considered evidence of health education having failed.

There is another side to the whole problem. Throughout childhood and adolescence, and probably even in adult life, a high percentage of ascorbic acid requirements are provided by fruit and vegetables, and particularly citrus fruit. These foods help to complete a balanced diet. From a number of superficial surveys, we have made it clear that a large amount of the knowledge about health matters generally and nutrition in particular held by the average family came originally from nurses at the baby health centres. The steps by which such knowledge became incorporated in dietary habits are clear; orange juice is necessary for the health of babies and so on to childhood and later, and the regular consumption of some fruit daily, including citrus fruit, becomes a habit. If ascorbic acid was added to milk preparations, the need for the baby health centre nurse to keep on reiterating the virtues of orange juice would be greatly reduced, and it would not be long before a generation of mothers would arrive who would not be convinced of the importance of orange juice in the diet of infants and children. This could ultimately have a serious effect upon the overall dietary pattern of the population.

The problem, then, resolves itself into the justification of a step which would be made necessary by poor mothercraft and defective health education procedures.

It is reasonable to assume that a mother who has failed to give her baby an ascorbic acid supplement will also be below the desirable level in other areas of mothercraft, the giving of ascorbic acid being a "token" of the quality of attention. Surely our aim is to raise the standard of mothercraft, and this calls for a full coverage of the mothers in the community by the health service. To resort to the addition of ascorbic acid to infant dried milk preparations is, as was said earlier, an admission of health education having failed.

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CONCEPTS OF SPEECH PRODUCTION IN CLEFT PALATE CASES AND A POSSIBLE TECHNICAL AID.[1]

By DAVID L. DEY,
Sydney.

SOME of you may remember that at our last annual meeting I described the palatal movements visible through an operative opening in the face of a man with a normal speech pattern. Once again, let me stress that the important features in the functional closure of the naso-pharynx appeared to be as follows: (i) that there is no evidence of a circular sphincteric action in the posterior constrictor of the pharynx; (ii) that a bunching of the nasal surface of the soft palate into a hillock is a constant feature during speech; (iii) that this hillock then moves up and down, with a relatively slow piston-like movement, the posterior surface making contact high up with the immobile posterior wall; (iv) that very rapid tongue movements are apparent; (v) that there is a marked inward movement of the lateral naso-pharyngeal walls, which is of importance in closure. Further to the above-mentioned if a large number of older children with repaired cleft palates are observed, it becomes apparent that the only constant feature in those with good speech is the presence of a freely mobile palate. In other words, the possession of a long palate does

[1] Read at a meeting of the Australian Paediatric Association, Canberra, April 17 to 20, 1959.

not appear to be of prime importance, and in fact, does not help unless the palate moves freely.

Similarly, most patients with poor speech (excluding those with grossly scarred or unsatisfactory soft palates) have a capacious pharynx, which is conspicuously larger than the normal. In spite of this, occasionally in a child with a large pharynx, in which complete physical occlusion appears anatomically impossible, good speech exists in the presence of an active palate. One is forced to the conclusion that complete occlusion is not essential, so long as the air current is sufficiently blocked or baffled.

With this thought in mind—the overwhelming importance of a loose palate which can rise sharply up into the naso-pharynx—I have compared the palate as it lies lax and loose at the end of operation with its appearance a few weeks later, when scar contracture has closed the lateral defects resulting from mobilization. At this later date the soft palate is relatively taut (as it remains), and it seemed worthwhile to attempt to obviate this change.

It is a basic principle of plastic surgery that contracture of the scar of a wound can be prevented with certainty only by filling the defect with normal tissue with its blood supply intact—that is, by means of a pedicled flap. The only immediately available source of such a flap in this area appears to be the mucosa of the cheek.

The operation to be described has resulted from these thoughts.

Operative Details.

A high standard of intratracheal anæsthesia is essential—blood transfusion is almost never necessary. Adequate exposure is obtained by means of a combination of Kilner's gag and Denis Browne's ring retractor, which pulls back the angles of the mouth. This allows the entry of sufficient light to make unnecessary the use of a headlight. The usual position is employed, with the patient on his back and his neck extended over a sandbag, and the operator standing at the head of the table.

The early part of the operation follows largely that described by Denis Browne. A long incision is made along the pterygo-mandibular raphe, and continues just within the alveolus to the canine tooth. The posterior part (pterygo-mandibular) is opened up by a palate elevator, and the anterior pillar of the fauces is separated from the mandible by a scissor cut.

The pterygoid hamulus is fractured and separated from its base. This step is essential if the tensor tendon is to be released, and allows the dissection to be carried up along the medial pterygoid plate. The muco-periosteum is elevated from the bone from before backwards, the posterior palatine vessels being ligated and divided, and the palatine aponeurosis being separated from the posterior border of the hard palate. The nasal mucosa is also freed from the bone of the hard palate.

Finally, the edges of the cleft are pared in the region of the uvula and split further forwards. The nasal surface is sutured with 40 "Mersilene", then the oral surface, and closure should prove to be quite free from tension. Care should also be taken to join muscle to muscle.

At this point the soft palate lies loosely in the mid-line, and there are two large lateral openings lying medial to the pterygo-mandibular raphe on each side.

A sharp hook is now inserted into he mucosa just posterior to the opening of the parotid duct, and traction is exerted forward. A second hook placed nearer the mandible helps to put the remainder of the mucosa on the stretch, and an incision is made from the mandible cranially upwards along the line of the hooks as high into the upper sulcus as possible. It then turns backwards along the upper sulcus to enter the residual lateral defect, and the flap outlined is raised, remaining attached at its lower border. Care should be taken that the bucco-pharyngeal fascia is not damaged, or the sucking pad of fat will prolapse into the mouth. However if it does occur this does not seem to matter greatly, as the pad of fat flattens down again as the wound heals. Some of the buccinator muscle is included in the flap, but the nerve supply of the remainder is not damaged and no change in function can be seen once the wound heals. Some bleeding may occur during dissection near the base of the flap, but has not proved troublesome. The pterygo-mandibular raphe is lifted with the flap.

Once mobilized, the flap is loose and elastic, and is easily rotated around the mandible and sewn into the primary lateral defect. It is of great importance to carry it down as far as possible, so as to bring it to lie between the soft palate and the bone of the mandible, in the region of the division of the anterior pillar of the fauces.

A defect remains on the cheek, but it heals rapidly, and is not visible a few weeks later. The additional operative time necessary is of the order of 20 minutes.

Comment.

This repair has been completed in 12 cases, and a year has passed since the period of the trial ended. In all, healing has been rapid and complete. It is, of course, far too early to draw any conclusions about speech in these children, none of whom has reached the age of three years. However, examination of their palates from the mouth has given cause to believe that the desired result has been achieved, at least in the majority of cases. Their palates appear loose and fleshy and free from lateral tension, while on phonation they move very freely up into the naso-pharynx.

Time alone will tell whether speech results will be better than those achieved by more conventional operations—whether a palate-lengthening operation is necessary in addition to the flaps, and whether the principle is applicable to patients operated on a long time previously with poor speech or whether greater hope lies in pharyngoplasty or posterior pharyngeal flap operations.

PRIMARY CONGENITAL ARACHNOID CYSTS.[1]

By M. SOFER SCHREIBER,

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CONGENITAL arachnoid cysts are divisible into primary congenital cysts, in which the cyst is a developmental arachnoid cyst and is the primary lesion, and secondary congenital cysts, in which the cyst is secondary to a primary congenital malformation.

The commonest variety of primary cyst is the superficial temporal arachnoid cyst. A large collection of either clear or yellow fluid is situated in the anterior portion of the middle fossa and sometimes the adjacent anterior fossa, opening up the Sylvian fissure and causing a gap between the frontal and temporal lobes. In well-marked cases the insula is uncovered, the free edge of the tentorium is often seen, and the internal carotid artery and its major branches, together with adjacent cranial nerves, are clearly visible without retraction of the brain. The outer wall of the cyst is arachnoid membrane, and in most cases there is an associated local bulging of the skull.

Very often this temporal bulge is the only symptom. Sometimes epilepsy is present, and sometimes there are symptoms of increase of intracranial pressure.

The deep temporal arachnoid cyst is rare. It appears to be an enormous cystic dilatation of a superior pocket of the cisterna ambiens, extending upwards between the insula and the inner aspect of the developing temporal lobe, pushing it laterally and thinning it down.

Equally rare is the interhemispheric arachnoid cyst, which lies between the posterior halves of the cerebral hemispheres on either side of the falx cerebri.

Congenital arachnoid cysts of the posterior fossa are more common; they compress the cerebellum and kink the aqueduct, causing hydrocephalus. These cysts are associated with cerebral abnormalities which date from about the fourth month of fœtal development, and the cysts are sited about large subarachnoid spaces—the cisterna magna, the cisterna ambiens and the cistern of the corpus callosum, and along the lateral fissure. These large subarachnoid spaces arise by a breaking down of a mesenchymal syncytium. An incomplete breakdown of

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trabeculæ may form a poorly-draining cystic pocket, which enlarges progressively because of the pulsatile thrusting force of the cerebro-spinal fluid.

These patients are in a precarious balance of intracranial pressure, and minor trauma may precipitate acute symptoms. When symptoms are present, the cyst should be drained into an adjacent cistern or ventricle.

IMPERFORATE ANUS.[1]

By F. DOUGLAS STEPHENS, M.S., F.R.A.C.S.,
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THE term "imperforate anus", as loosely used by many clinicians, includes several developmental anomalies, and takes no cognizance of whether the bowel terminates blindly, or communicates with the urinary or genital tracts or with the skin of the perineum. In this paper malformations of communicating type are excluded from the discussion.

When the bowel terminates blindly, special techniques are necessary to determine the level at which the rectum lies. On the other hand, if a genital, urinary or cutaneous communication exists, the site of the orifice of the rectal or anal communication gives the clue to the level at which the patent bowel lies.

In male infants, the levels of the normal calibre rectum, in the blind and communicating anomalies, correspond closely. In females, blind anomalies are rare, whilst communicating anomalies are transposed by the Müllerian migration to variable levels.

The object of investigation of these anomalies is to determine accurately the furthermost point in the pelvis to which the normal calibre bowel descends. The pathological anatomy of the sphincters corresponding with the determined levels can then be defined. Upon this information, a rational surgical procedure may be adopted with the purpose of constructing a sphincteric mechanism for the new anal canal.

Clinical investigations of male infants reveal that the rectum ends at one of three levels, all of which are regarded as of embryological significance (Figure I).

In this paper, the development and diagnosis of imperforate rectal and anal anomalies at these three levels in male patients are studied. Only the management of the highest level anomaly, the most difficult of all, is considered in any detail.

Embryology.

The rectum and anal canal are developed in three subdivisions—namely, the hind gut, the cloaca and the anus. The sites of junction of these three contributions account for the constant appearance of embryological abnormalities at three levels (Figure I). It is also at these three levels that communications with the urinary or genital tracts or the skin occur.

"Imperforate rectum" in this paper denotes the presence of the short hind-gut segment only, and the absence of the cloacal and anal contributions. "Imperforate anus" denotes the presence of the cloacal and hind-gut contributions and the absence of the anal pit. "Imperforate anal membrane" denotes the lowest abnormality, in which all contributions are present, but the anal membrane between the cloacal and anal zones has failed to break down.

Radiographic Diagnosis of the Level.

The Wangensteen-Rice (1930) inversion technique of demonstrating the gas-filled terminal portion of the bowel was used in the neonatal period.

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

The level of the gas shadow may be assessed in relation to a marker on the skin of the perineum, or, more accurately and with advantage, to the bony landmarks of the infant pelvis. These levels are determined from studies of the lateral views of the pelvis (Figure II).

The pubo-coccygeal line is one important landmark; it begins at the ossification centres of the pubes, and crosses

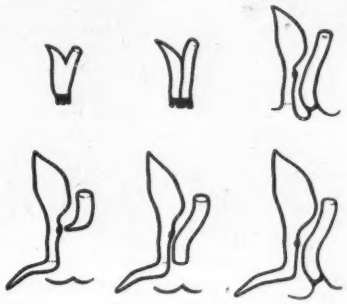

FIGURE I.

Upper row: three stages in development of the rectum and anus; lower row: deformities associated with arrest of development at each stage—imperforate rectum, imperforate anus and imperforate anal membrane.

the pelvis to the sacro-coccygeal junction (Stephens, 1953). This line between two fixed points in the new-born pelvis represents the lowest level to which the hind-gut descends in the imperforate rectum deformity.

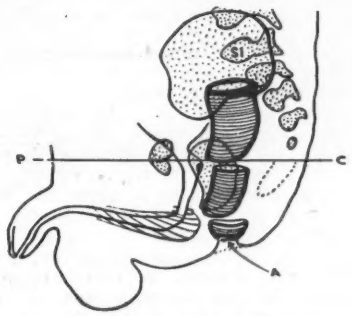

FIGURE II.

Level of deformities in relation to bony landmarks. P-C, the pubo-coccygeal line. A denotes the anal membrane. Imperforate rectum: the terminal portion of the perforate bowel extends to the pubo-coccygeal line or more cranially. Imperforate anus: the cloacal contribution extends to the lower margin of the ischial bone. Imperforate anal membrane: the cloacal contribution in contact with the anal pit migrates to a point approximately 1·5 cm. caudal to the ischial bone.

The outline of the ossified ischium is the second important landmark (Figure II). Here it is important to make certain that the two ischial bones lie one directly over the other. This is ensured by accurate centring of both the cone of the X-ray machine and the greater trochanter of the patient on the middle of the film. To assist the radiographer, mark the skin over the greater trochanters clearly and boldly with a cross.

The cranial end of the shield-shaped ossified ischium in the new-born is cut across by the pubo-coccygeal line and represents the common level of the terminal portion of the bowel in imperforate rectum deformities. The caudal

end of the ischium is the level to which the rectum descends in the malformation of imperforate anus.

In the event of absence of lower sacral vertebræ, the *"pubo-coccygeal" line should run from* the symphysis pubis across the pelvis, cutting through the cranial lip of the ossified ischium.

Sometimes difficulties are experienced in determining the level of the terminal portion of the bowel by the *Wangensteen and Rice technique.* Then a timely transverse colostomy will serve a dual purpose. It will overcome the obstructive crisis and provide a route of access *for deliberate "Lipiodol" studies of the terminal portion* of the bowel.

The three levels can now be related to these fixed bony landmarks. Firstly, the bowel in imperforate rectum extends to the pubo-coccygeal line. Secondly, the bowel *in imperforate anus reaches to the lower margin of the* ischial bone. Thirdly, the imperforate anal membrane lies approximately 1 to 2 cm. caudal to the lower margin *of the ischial bone.*

Sphincter Muscles.

The three main instruments of continence in normal humans are the internal and external sphincters and the pubo-rectalis part of the levator ani muscles.

The developmental visceral anomalies of the anus and rectum under discussion are accompanied by variations *in development of these sphincter muscles.* In all three, the internal and external sphincters are rudimentary (Stephens, 1953).

In the two upper level anomalies, the pubo-visceral sling of the levator ani muscle is the muscle which is both *available and amenable for corrective surgery.* This muscle, if properly used, is capable of furnishing a high degree of continence. It is the presence of this muscle *which justifies surgical reconstruction of the rectum and* anal canal.

Imperforate Rectum.

In this abnormality, the internal and external sphincters are absent or so rudimentary that they are of no surgical *import in reconstructive operations.*

The levator ani musculature is well developed, though abnormal. The pubo-rectalis sling, lying caudal to the rectum, blends with the pubo-urethralis sling. It is a rule that, in the absence of the cloacal contribution of the *rectum, the pubo-rectalis muscle surrounds the urethra* or cloacal canal in the male or, in the female, the cloacal canal or vagina.

That part of the levator ani muscle which constitutes the pubo-urethralis muscle, in this anomaly, is the muscular collar at the anterior end of the pelvic diaphragm. The collar is attached to the pubis anteriorly and surrounds the urethra, being attached caudally to the perineal body. It has an intimate relationship to the posterior wall of the urethra as high up as the verumontanum or pubo-coccygeal line, and is closely adherent to the caudal aspect of the terminal portion of the recto-urethral fistula.

Then the muscle of the collar merges into the muscle of the pelvic diaphragm, which lies on a plane at right angles to that of the collar.

The terminal portion of the rectum is cranial and posterior to this collar and on the cranial aspect of the diaphragm.

In this group of anomalies, partial or complete sacral agenesis may coexist. Then the neural outflow to the levator ani (second, third and fourth sacral nerves) may be impaired. This is regarded as inevitable when there is also evidence of impairment of the vesico-urethral function.

Imperforate Anus.

Here the internal sphincter is absent and the external sphincter is rudimentary. The pubo-rectalis collar forms a hammock-like encirclement of the rectum. The rounded terminal portion of the bowel is attached to and "sits" in the hollow of the hammock.

The diaphragmatic portion of the levator ani muscle is *complete and sound.*

Imperforate Anal Membrane.

In this abnormality — the lowest of the three — the internal sphincter, which normally lies below the level of the valves, is absent. The external sphincter is represented only by its deepest portion, which forms an effective sphincteric ring above the level of the membrane.

The pubo-rectalis muscle encircles the bowel, gaining intimate attachment to it at the imperforate membrane.

Both the inner circular and outer longitudinal smooth- muscle coats of the rectum end in a fibro-muscular knot at the lowermost point of the blind terminal portion of *the bowel on the membrane.*

In all three abnormalities, it is apparent that the *sphincters are radically different from normal.* In the high and middle levels, the only sphincteric muscle available for use in surgical construction of the anal canal is *levator ani musculature. With the low-level imperforate* membrane, the deep part of the external sphincter is present in addition. In all, the internal involuntary *sphincter as described in the normal child is non-existent.*

Surgical Management.

The chief surgical target is sphincteric control over the reconstructed anal canal, for it is better to provide a *patient with a permanent abdominal colostomy than with* a totally incontinent perineal anus.

The one muscle which is present in all three anomalies is the levator ani, and this muscle, if appropriately used in reconstructive operations, gives a satisfactory degree *of continence of fæces.*

Relatively simple procedures will be adequate for the *imperforate anus and the imperforate anal membrane.* These operations may be performed in the neonatal period through carefully planned perineal incisions.

The imperforate rectum or high-level anomaly requires more complex operations. In this paper, only the treat*ment of this problem will be discussed.* The basic abnormality of the levator ani and its pubo-urethralis division must be carefully handled to ensure maximum sphincteric advantage to the newly constructed anal canal. A false move here means the difference between a very good and a poor result (Figure III). On the one hand, the baby is immediately continent; on the other hand, he is incontinent of fæces and soils for many years, and at best he may develop a *minimum, though adequate, degree* of continence when he is capable of bringing all his intelligence to bear.

Surgeons are divided in their opinions regarding, firstly, the age at which to perform the definitive operation, and secondly, the route of access to the field of operation. Here a temporary defunctioning transverse colostomy is favoured. It is important to wash out the meconium from the distal blind end of the rectum when a urinary fistula coexists. Subsequent reconstructive surgery is then performed when the pelvic dimensions have enlarged, preferably not before the age of one year.

The first step in the operation, and the one that determines the route of approach, is the canalization and enlargement of the pubo-urethralis muscle in such a way that it will accommodate the new recto-anal canal snugly within its sling-like structure (Figure III).

This sling hugs so closely and so intimately the urethral wall, which, in turn, lies against the symphysis pubis, that it is safer to approach it from a posterior aspect than from the abdominal route. The sacro-coccygeal route, though confined, affords this access to the vital sling, even though the muscle cannot be actually visualized. The supra-levator space can be cleared to the urethral wall, which is easily identified by the aid of a sound or firm catheter in the lumen. The angled forceps are introduced so that their tips turn caudally, clinging closely to the posterior wall of the urethra and to the mid-line of the pelvis. Inevitably, the sling of the levator will be

ILLUSTRATIONS TO THE ARTICLE BY M. L. POWELL.

FIGURE I.—Female patient, aged 20 months, grossly disabled. The right ventricular pressure was 100 mm. of mercury, and ear oximetry produced a figure of 84%. The cardiac contour was as shown in Figure VII. Central cyanosis was present, also a degree of peripheral stagnant anoxia due to poor cardiac output.
FIGURE II.—The patient shown in Figure I, after operation. Ear oximetry resulted in a figure of 96%. Great symptomatic improvement was evident.

FIGURE III.—Hands of patient in Figure I, showing gross clubbing before operation. FIGURE IV.—Hands of patient in Figure I, after operation, showing improvement.

ILLUSTRATIONS TO THE ARTICLE BY M. L. POWELL.

FIGURE V.—X-ray film of mildly affected subject, symptomless, with right ventricular pressure of 40 mm. of mercury, but with well marked post-stenotic dilatation. FIGURE VI.—Angiogram of subject in Figure V, showing post-stenotic dilatation. FIGURE VII.—X-ray film of patient in Figure I, showing cardiac enlargement, with post-stenotic dilatation which pulsates forcibly under the screen whilst the lung fields are "quiet" and oligæmic. This was one of the cases in which, at operation, a gross systolic thrill was present over the right atrium, due to tricuspid incompetence. This thrill vanished immediately after valvotomy.

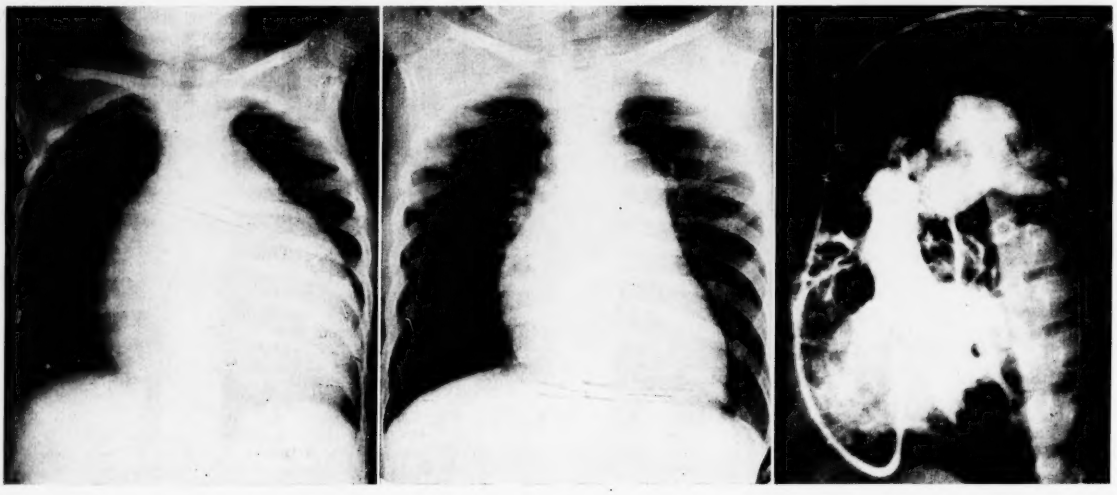

FIGURE VIII.—The patient was a boy, aged eight years, grossly disabled and with increasing cyanosis. The right ventricular pressure was 135 mm. of mercury, and the main pulmonary artery pressure was 5 mm. of mercury. The heart was huge, and the post-stenotic dilatation was submerged in the cardiac mass. FIGURE IX.—Patient in Figure VIII, 12 months after operation, showing great reduction in the size of the heart. The post-stenotic dilatation is now strikingly clear. FIGURE X.—The selective angiocardiogram of the patient in Figure I, showing the unobstructed infundibulum, the outline of the thickened stenotic valve, the jet passing through and the gross post-stenotic dilatation.

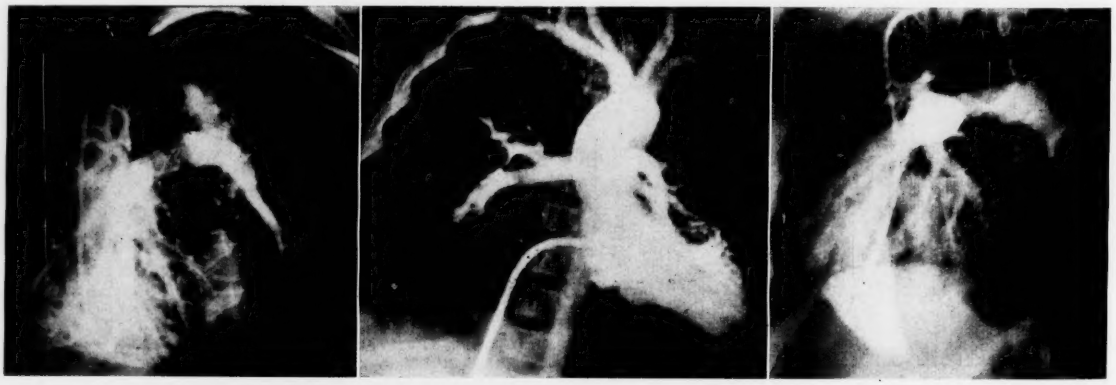

FIGURE XI.—Angiocardiogram of a patient with tetrad of Fallot, showing the aorta filling simultaneously with the pulmonary artery, without the opaque material passing from the right to the left atrium; this could occur in pure valve stenosis with tricuspid incompetence. Left anterior oblique view. FIGURE XII.—Antero-posterior view of the patient in Figure XI. FIGURE XIII.—Angiocardiogram in a case of tetrad of Fallot. Though the aorta has apparently not filled, a tell-tale puff of opaque material in the left ventricle shows that a ventricular septal defect is in fact present.

canalized in this way. The sling is then enlarged sufficiently to accommodate the new anal canal with some tightness (Figure III).

In some infants the whole operation can be performed through a combined sacro-coccygeal and perineal approach; in others, especially those in whom the hind-gut terminates at levels higher than the pubo-coccygeal line, the sacro-coccygeal "canalization" procedure must be combined with abdominal mobilization to procure the necessary length of bowel required to reach to the perineum.

Sphincter Control.

In the deformities imperforate anus and imperforate anal membrane, the pubo-rectalis muscle, being already anatomically poised for sphincter action, suffices for a satisfactory state of continence once the new anal canal is constructed. With the imperforate rectum deformities, however, the pubo-urethralis muscle must be converted surgically into a pubo-rectal sling. If this step is satisfactorily executed, then satisfactory continence of fæces is assured.

Post-operative assessment by digital examination will reveal the presence of a good sphincteric sling operating high in the pelvis in the successful cases (Figure III). Continence is established within a few weeks.

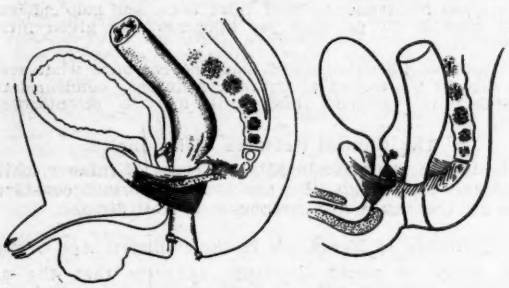

FIGURE III.

Two diagrams to show the correct (left-hand diagram) and the incorrect relation of the pubo-rectal sling to the newly constructed anal canal.

If the pubo-urethralis sling is damaged in the operative procedure or is ignored (Figure III), then digital examination may detect only a weak lateral pinching action by the non-sphincteric fibres of the levator diaphragm. Some degree of continence may be acquired when the intelligent child associates anal control with "levator squeeze".

Conclusions.

Rectal and anal deformities which cause total occlusion pose special problems in diagnosis and management. Three such visceral abnormalities in the male are the imperforate rectum, imperforate anus and imperforate anal membrane.

The levels of occlusion of all three can be identified radiographically by comparing the gas shadows in the terminal portion of the bowel with fixed bony landmarks of the pelvis.

The pubo-coccygeal line denotes the level of the imperforate rectum, the lower border of the ossified ischium denotes the level of the imperforate anus, and a point approximately 1·0 to 2·0 cm. caudal to the ischium is the level of the imperforate anal membrane.

For each of these visceral deformities, there are consistent associated skeletal anomalies—namely, a rearrangement of the levator ani musculature and rudimentary sphincters.

Having diagnosed the visercal deformity and knowing the skeletal anatomy conforming to the anomaly, the surgeon can reconstruct the anal canal, using to maximum advantage the pubo-visceral sling of the levator ani. In all three deformities, the newly constituted pubo-rectalis muscle is the chief guardian of continence.

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STUDIES OF NORMAL AND ABNORMAL URETERAL PERISTALSIS.[1]

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AND

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THE role of mechanical obstruction in urinary infection is widely appreciated, and our routine techniques of examining the urinary system are designed to detect mechanical defects. However, as more careful investigations are made of children's urinary systems, it becomes apparent that functional defects are frequently the cause of infection and destruction of renal tissue. A functional unit of the system which rarely is studied as a routine is the ureter. I should like to present the results of our efforts to detect defects in this part of the urinary transport system. Monometric tracings of ureteral peristalsis have been made in 231 cases. In a number of these cinefluorographic studies were also made. To assure an adequate flow of urine, an intravenous infusion was administered during the studies. Of the 231 patients, 130 were classed as having normal peristaltic function. These had contractures occurring at intervals of 7 to 20 seconds. The average peristaltic wave developed 15 cm. of water pressure in the upper part of the ureter, 25 cm. in the middle portion and 35 cm. in the lower part of the ureter. Peristaltic waves of this intensity are needed to overcome the uretero-vesical resistance, which is about 15 cm. It is also required to force the urine into the bladder, particularly during the later stages of filling.

There were 101 children classed as having abnormal peristaltic function. Infrequency of contractures, so that they occurred at one-minute intervals, was considered abnormal. Such slowing was rare except when associated with diminished amplitude of contractures. A pressure of less than 20 cm. of water was considered abnormal.

The causes of this inadequate function can be placed in three categories. The role of infection has fairly well been established, for we have observed instances in which eradication of chronic infection has been accompanied by an improvement in ureteral function or even by return of normal contractions.

Chronic obstruction and dilatation of the ureter may lead to diminished peristalsis. Evidence for this has been gained from experimental procedures, in which chronic ureteral obstruction has been produced and repeated recordings of ureteral peristalsis have been made. Whether release of obstruction is followed by a return of peristalsis is not definitely known. Probably the degree of ureteral enlargement may determine whether this is possible or not.

Congenital defects in peristalsis have been observed, particularly in new-born infants, when there has been neither infection nor mechanical obstruction.

Cinefluorographic studies of the ureter may present misleading information. There may be changes in contours which appear to be effective pumping action. By recording simultaneous pressure tracings and cinefluorographic studies, it becomes apparent that these changes

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

in contour may produce no or minimal changes in intraluminal pressure, and hence no transport of urine is accomplished. The apparent reason for this is that the contracture fails to occlude the lumen, so that the fluid escapes up the ureter rather than being forced into the bladder. A normal contracture occludes the ureter and forces the urine before it, leaving the ureter empty behind the contraction wave.

Mistakes in interpretation of cinefluorographic studies can be avoided by observing whether fluid is forced into the bladder. This can be accomplished by making observation when the bladder is empty of contrast medium.

The practical value of peristaltic studies is that in obscure cases of infection this examination may offer an explanation for the child's illness. It also gives additional evidence of the importance of eradication of infection.

In certain conditions effective peristalsis is of extreme importance. For instance, when there is ureteral reflux and reimplantation is considered, the presence of peristaltis is essential if the reimplantation is to be successful. When there is a massive anomaly of the urinary system, such as exstrophy of the bladder, reconstruction is unwise unless peristaltic function is present.

It is apparent that in the understanding of the cause of infection in obscure cases and in evaluation of patients for various surgical procedures, ureteral peristalsis is important.

NEONATAL MENINGITIS.[1]

By ELIZABETH K. TURNER, M.D.,
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IT IS proposed to discuss the subject of neonatal meningitis under two sections: (i) the presentation of an unusual case of neonatal meningitis; (ii) a brief résumé of the chief clinical features of the disease, my facts being gleaned from world literature and from a study of 45 case histories of infants who contracted the disease within the first 28 days of their existence, and who were treated at two Melbourne hospitals during the last five years.

Report of a Case.

Baby G., a female, was born at the Queen Victoria Hospital, Melbourne, on November 27, 1958. Her presenting symptom was icterus gravis; the provisional diagnosis made was erythroblastosis fœtalis due to an ABO incompatibility between the maternal and fœtal blood groups; but for the lumbar puncture findings, meningitis would probably never have been suspected in this infant. The infecting organism also was not one of the usual enteric group, but a fairly normal inhabitant of the maternal vagina, and one which is usually of low-grade pathogenicity. The infant was the result of the fifth pregnancy of Mrs. G., who was aged thirty years and of blood group O, Rh-positive. Her serum contained a low titre of anti-A and anti-B agglutinins throughout the pregnancy.

Mr. G. was healthy, and his blood group was B(3) homozygous Rh-positive.

Throughout the pregnancy, Mrs. G. was said to have "sinusitis", with a clear nasal discharge. At eight weeks' gestation she developed a yellowish, non-specific vaginal discharge, which had disappeared at term. At 24 weeks' gestation she developed a "boil" in each nostril and pimples on the face; these were treated with antibiotic ointment and sulphonamide given by mouth. At 36 weeks' gestation she developed typical rubella, and was in bed for seven days. Twenty-two days before delivery she had a small hæmorrhage of dark blood, and occasional "spotting" continued until delivery. Her total weight gain throughout pregnancy was 23 pounds, and she seemed well at term. She was delivered spontaneously at term of a normal-looking female infant. The membranes ruptured 17 minutes before the birth of the child, and the liquor was noted to be meconium-stained. No vaginal examination was performed at any time. The infant breathed immediately; her weight was 3·27 kg. (7 lb. 2·75 oz.), and she measured 48·75 cm. (19·5 in.).

At the age of 14 hours jaundice was noticed, and the infant became progressively less active. Her blood group was B(3) Rh-positive, and at 40 hours the serum bilirubin content was found to be 18 mg. per 100 ml. The positive signs at this time were as follows. Deep jaundice and a frown were present; the infant resented movement and showed increased "startle" response if disturbed; her cry was shrill, she was sucking poorly, and the fontanelle felt woody. A number of expected signs were absent—there was no neck stiffness, no vomiting, no fever and, in spite of the deep jaundice, no hepatosplenomegaly. A lumbar puncture revealed turbid cerebro-spinal fluid, 6 ml. being removed; this contained 7300 polymorphs per cubic millimetre, and on culture a streptococcus group B, weakly hæmolytic was grown. This streptococcus was identical with one cultivated from the mother's post-partum cervical swab. There had been little contact between the mother and her offspring after birth; the mother's nose and throat swabs did not contain the streptococcus, and thus it was probably a normal inhabitant of her vagina. No streptococci were grown from nasal or umbilical swabs of the infant, but the group B streptococcus was isolated from her throat swab, in association with a coagulase-positive staphylococcus.

Vigorous treatment was commenced at the age of 41 hours. Chloramphenicol was given intravenously in doses of 100 mg. per kilogram, 1,000,000 units of crystalline penicillin were given intravenously ever third hour, and sulphadiazine in a dose of 0·1 gramme per kilogram was given intramuscularly.

In spite of this, the infant made no response whatsoever and died at the age of 63 hours. An autopsy confirmed the diagnosis of purulent meningitis due to streptococcus group B.

The Clinical Features of Meningitis.

Meningitis is predominantly a disease of infancy, childhood and early adult life; age is an important consideration in the cause and prognosis of the disease.

Age Incidence of Meningitis in the Pædiatric Age Groups.

A study of world literature suggests that the age incidence of meningitis in pædiatric age groups follows the following pattern:

Birth to One Month:

Approximately one-quarter of all pædiatric cases occur in this group; almost 30% of these are in premature infants. *Hæmophilus influenzæ*, the meningococcus and the pneumococcus are rare.

One to Two Months:

This is the peak incidence of bacterial meningitis; approximately half of all pædiatric cases occur in this group. *H. influenzæ* is the most common single cause.

Two to Three Years:

There is a high incidence of *H. influenzæ*, meningococcal, pneumococcal and tuberculous meningitis in this period.

Three to Ten Years:

The incidence of *H. influenzæ* meningitis declines markedly in this period. Pneumococcal, meningococcal and tuberculous meningitis decline more slowly; more than half of all these cases occur before the age of 10 years.

Ten to Twenty Years:

There is a high incidence of viral meningo-encephalitis—for example, mumps, herpes, poliomyelitis, Coxsackie infection, E.C.H.O. infection and leptospirosis in this period. Three-quarters of all these cases occur before the age of 20 years.

Sex Distribution.

In world literature, meningitis appears to be a disease of males, the incidence for all age groups being of the order of 2:1. In my series there were 21 female infants and 23 male infants.

The Pathogenesis of the Disease.

The pathogenesis of the disease is set out in Table I.

The predominant microorganisms found in the meninges in cases of neonatal meningitis are those of the enteric group, including coliform organisms, paracolon bacilli,

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

TABLE I.
Infecting Organisms in Neonatal Meningitis.[1]

Source.	Enteric Organisms.			Staphylococcus.	Streptococcus.	Pneumococcus.	Other and Unknown.	Total.
	Escherichia Coli.	Paracolon Bacillus.	Other Coliform Bacilli.					
Watson (1957)	14	10	9	1	6	1	2	45
Smith (1955)	13	2	1	—	—	1	—	17
Flensborg (1943)	48	—	20	20[2]	32[2]	12[2]	8	131
Beveridge (1954)	7	—	3	—	1	4	1	16
Debré and Mozziconacci (1949)	17	—	8	3	4	7	7	46
Zlai and Haggerty (1958)	25	2	12	12	15	7	7	83
Present series	21	2	2	3[2] 3	5	6	6 (including 2 *H. influenzæ* infections)	45

[1] Adapted from Watson (1957), Table II, with the addition of 83 cases from Zaia and Haggerty (1958) and 45 cases in the present Melbourne series.
[2] Some in mixed infections.

Proteus, Salmonella, Listeria, Pseudomonas and *Alcaligenes fæcalis.* These organisms occur in approximately 55% of all cases. The reason for their predominance is unknown; but it has been found (by Wright, H. P., cited by Randall, 1948), that although there was a passive transfer of maternal antibodies to the fœtus, particularly in relation to *H. influenzæ*, pneumococci and meningococci (if the mother was immune), there was no one mother of 55 tested who had agglutinins to all seven types of coliform organisms tested. Thus appreciable resistance does not seem to be transferred against organisms of the coliform group. Furthermore, Flensborg (1943) suggested that the increased permeability of the neonatal gastro-intestinal mucosa was an important pathogenic factor in the development of meningitis, as he noted that in all his cases due to coliform organisms bacteriæmia was present.

Coliform bacteriæmia is a well-known phenomenon in calves, and probably frequently occurs in human infants.

It is now a well-established fact that, during maternal bacteriæmia (or viræmia), organisms may traverse the placenta and cause infection in the fœtus—for example, in hæmatogenous tuberculosis or in the meningo-encephalitis of the new-born due to Coxsackie B virus, which has been reported in nursery epidemics associated with aseptic myocarditis (Kilbrick *et alii*, 1956).

Routes of Infection of the Meninges.

Intrauterine infection may occur transplacentally from the mother's blood, or from an abdominal source via the Fallopian tubes, or infection may occur in the birth canal, whether the membranes are ruptured or not. Extra-uterine infection can occur by any of the usual routes during the neonatal period.

Predisposing Causes.

Premature infants seem particularly susceptible to infection. It is suggested that the known increased permeability of their blood-brain barrier is a factor in the pathogenesis of this disease (Orlandini *et alii*, 1955), although it was also demonstrated (Norton *et alii*, 1952), that total gamma globulin and properdin levels are not so low in the new-born as they are in the second and third months of life. Gluck and Silverman (1943) have shown that the leucocytes of premature infants have decreased phagocytic ability, which may also be a factor in their susceptibility to any infection.

Perinatal abnormalities, including early rupture of the membranes, forceps delivery, intracranial hæmorrhage or œdema and cephalhæmatoma, seem to predispose to the development of meningitis, possibly by alteration of local tissue resistance to infection, associated with increased blood supply and œdema of the cranial tissues. In the Boston series (Zlai *et alii*, 1958), perinatal abnormalities were present in 19 of the 83 cases; in my series in 11 of the 45.

Sepsis, including umbilical infection, skin sepsis and conjunctivitis, may be a predisposing cause. Such a state was present in at least seven infants in my series, and another eight infants had pneumonia primarily. Otitis, which is extremely rare in the perinatal period, was present in two infants in the series prior to the onset of meningitis.

Symptoms and Signs.

The symptoms and signs of neonatal meninigitis are notoriously conspicuous by their lack of specificity, the most commonly encountered being poor activity and abnormal feeding. The symptoms, in order of their frequency and importance, are as follows: (i) poor activity (pallor, frown, poor general appearance); (ii) abnormal feeding (vomiting or poor sucking); (iii) irritability and, if disturbed, attacks of "startle"; high-pitched cry; (iv) respiratory distress (cyanosis); (v) full (or woody) fontanelle; (vi) fever (in half the number of cases only); (vii) convulsions (in less than half the number); (viii) jaundice; (ix) diarrhœa; (x) neck stiffness.

Prognosis.

The prognosis is generally grave. This is often a mortal infection, in spite of what would have been ordinarily considered adequate therapy, and in spite of early diagnosis.

Of the Boston series of 83 infants, 62 died and 21 survived; in the present Melbourne series, 28 infants died and 16 lived.

Summary and Conclusions.

Meningitis is not uncommon during the neonatal period, and carries with it a grave prognosis. There is a mortality rate of about 60% to 70%. The predominant infecting organisms are members of the enteric group.

Reasons are suggested for the high incidence of meningitis in the neonate.

The histories of 45 infants suffering from neonatal meningitis in two hospitals in Melbourne have been reviewed; 28 infants succumbed to their infection.

The presenting signs and symptoms are notorious for their lack of specificity.

A case is presented of an infant who died at the age of 63 hours (in spite of vigorous therapy) of meningitis due to a weakly hæmolytic Group B streptococcus. This organism was identical with one cultured from the mother's cervix *post partum*. Infection had evidently been acquired during the infant's passage through the birth canal, in spite of the fact that there was no perinatal trauma.

This infant's predominant symptom was jaundice, and at first a diagnosis of fœtal erythroblastosis was entertained.

The case is noteworthy, in that a relatively innocuous organism inhabiting the birth canal could have caused such fulminating and rapidly fatal infection in the offspring.

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DIAGNOSIS OF ENTEROVIRUS INFECTIONS.[1]

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A STUDY of patients admitted and enteroviruses (poliomyelitis, E.C.H.O. and Coxsackie viruses) isolated at Fairfield Hospital during the period January, 1954, to February, 1959, demonstrates some of the difficulties entailed in correlating virus isolation with disease.

The majority of enteroviruses isolated in this series have been derived from the fæces of patients with aseptic meningitis; but a proportion has been associated with conditions as diverse as whooping-cough, Stevens-Johnson syndrome, bacterial meningitis and pyrexias of unknown origin. E.C.H.O. and Coxsackie viruses have been isolated from fæces of patients with paralytic poliomyelitis.

It has been demonstrated by Dr. A. A. Ferris that enteroviruses can be isolated in large numbers from "normal" children. It has been said by Dr. Huebner, of similar findings, that "no one has yet reported a virus responsible for good health".

Koch's postulates have provided a traditional didactic guide in the bacterial field, but these are not wholly applicable to the characteristics of natural virus infections or to the techniques of their culture. There is little doubt that Koch would have denied the causal relationship between poliovirus and poliomyelitis and even, probably, the pathogenicity of this virus.

Rivers modified Koch's postulates in 1937, and 20 years later Huebner advanced additional criteria based on modern techniques. These criteria advanced by Huebner are designed to define and establish a virus as a potential pathogen, and are only of partial assistance in assessing the ætiology in a particular case.

The other aspect important to the clinician concerns criteria to discern the causal relationship between a virus isolated from a patient and a coexisting disease.

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

Difficulties of Interpretation.

The interpretation of virus isolation in a patient is made more difficult by the following factors.

Persons infected with enterovirus may continue to excrete virus for periods of two months or more. Furthermore, symptoms appear in only a certain proportion of people infected, so that coincidental infection is quite likely.

The infections are widespread, and since the rise in antibody has often occurred by the time the patient presents with symptoms, this technique is robbed of its usual value in the diagnosis of infection.

Certain types of Coxsackie and E.C.H.O. viruses may be cultured frequently from the cerebro-spinal fluid of patients with meningitis, but this is not the case with poliovirus in meningeal poliomyelitis.

Enteroviruses are seldom isolated from the blood of patients.

Dual infection may complicate the interpretation of fæcal isolations. In this series, isolation of two enteroviruses from a single patient has occurred in seven instances, in some of which the verification of poliomyelitis infection was important.

Factors in Diagnosis.

The problem of diagnosis in humans is eased by concentrating a number of patients with these diseases, as happens at Fairfield Hospital. This permits a comparison of clinical and laboratory findings and the determination of epidemic viruses in relation to syndromes.

It can be shown that the fæcal isolation of these various enteric viruses is more frequent in these cases than in the rest of the hospital population.

By surveying the types of virus which have been isolated from internal sources, mainly cerebro-spinal fluid, and assuming that such an isolation is diagnostic, it is apparent from work done in this series alone that the following virus types are capable of producing meningitis (these viruses have all been isolated from the cerebro-spinal fluid of patients with meningitis): (i) E.C.H.O., Types 1, 2, 3, 4, 5, 9, 13 and 14; (ii) Coxsackie, Group A, Type 9, and Group B, Types 1, 2, 3, 4 and 5.

In the case of poliomyelitis, isolations of all three types have been made from various parts of the central nervous system at autopsy.

Such isolations from an internal source imbue with greater significance concurrent fæcal isolations during an epidemic period, and histories of contact with such cases can provide confirmatory evidence.

In this series, and also in the literature, there is no convincing evidence as yet that paralysis of the type seen in poliomyelitis is caused by either E.C.H.O. or Coxsackie virus infections.

In these cases, transient mild weakness evidenced by diplopia and occasional limb weakness not always unrelated to pain has been observed, but in no case has permanent weakness resulted in a patient with proven E.C.H.O. or Coxsackie infection.

Accumulating evidence from experimental work in animals and human volunteers, and antibody studies combined with epidemic observations, have led to the appreciation of certain reasonably well-defined clinical syndromes, which may substantiate the isolation of a virus from an external source, such as fæces, as the causal organism. For instance, the fæcal isolation of poliovirus from a patient with meningitis and persistent lower motor neuron paralysis is acceptable as diagnostic proof of poliomyelitis. Similarly, the isolation of a Coxsackie B virus from a patient with muscle pain and pleuritic type pain indicates a strong causal relationship. On the other hand, isolation of an E.C.H.O. or Coxsackie virus from a patient with a meningeal illness associated with persistent lower motor neuron paralysis would not alter the diagnosis of poliomyelitis.

FATAL COLLAPSE DURING EXCHANGE TRANSFUSION.[1]

By S. E. J. ROBERTSON,
Sydney.

SINCE 1952 I have carried out or personally supervised 452 exchange transfusions. On 19 occasions collapse occurred during the procedure, with death following within a few minutes to several hours. Autopsy was performed on 17 of these patients. In six subjects a cause for the death was found which was unrelated to the procedure and which would have been inevitably fatal. Briefly, there were a large intracranial hæmorrhage in one, a large mediastinal hæmorrhage in another, and in the remaining four a marked degree of hydrops fœtalis. These last four were moribund when the procedure was commenced. In the remaining 11, no changes were found except those of erythroblastosis fœtalis, apart from one subject who had acute pulmonary œdema.

Collapse occurring during exchange transfusion has been mentioned in various reviews of the subject. Walker and Nelingan (1955) stated that deterioration occurred during 40% of their exchanges and was usually due to overloading or to a type of shock of doubtful ætiology.

Allen and Diamond (1958), in their fine review of erythroblastosis fœtalis, detail many of the precautions to prevent collapse, and conclude that experience of the procedure is valuable, and that minor and intangible things may be of critical importance in some cases.

The possible causes of collapse will now be dealt with briefly.

Electrolyte Disturbances.

Electrolyte disturbances are thought to be hyperpotassæmia, hypercitræmia and hypocalcæmia. These changes were investigated by Farquhar and Smith (1958), who could find no consistent relationship between these changes and the occurrence of collapse. Further studies of a similar type are needed, particularly on the effect of excessive citrate levels. In a small series of patients at the Royal Alexandra Hospital for Children, no excessive rise of potassium level was found when blood was used which had been collected within three days. The dangers of hypocalcæmia have probably been exaggerated (Walker and Neligan, 1955).

Cooling.

It is usual for the rectal temperature of an infant undergoing exchange transfusion to fall to 96° or 97° F. If blood is injected rapidly inadequately warmed in a large amount relative to the blood volume, the effect on the myocardium may be profound. Pew (1955) described a case in which cardiac massage was necessary for cardiac arrest. Normal rhythm was restored, and the exchange continued with the heart under direct vision. He noted that the heart slowed and felt cooler while blood was being injected.

Overloading of the Circulation.

Overloading of the circulation is a very real danger to the severely anæmic patient who receives an exchange transfusion soon after delivery. These patients, probably owing to their anæmia, have a weakened myocardium, and have varying degrees of congestive cardiac failure, increased venous pressure and increased blood volume. It is necessary to reduce their blood volume as quickly as possible. Overloading can also occur when an error is made in measuring the amount removed and the amount injected.

One of the deaths in this series due to the exchange alone was from this cause. This infant, perfectly well apart from hyperbilirubinæmia, was having a third exchange. The method used was that described by Wiener (1950), in which blood is injected into the saphenous vein at the ankle and removed by bleeding from the radial artery. This is a very good, and usually safe, method; but its weakness is in accurate measurement of blood squirting or dripping from the wrist. On this occasion, the operator at the wrist was removing less than he thought he was, so that an excess was injected at the ankle. After about 150 ml. more had been injected than had been removed, the infant became violently restless and cyanosed, coughed up blood-stained fluid and collapsed. Coarse crepitations were heard all over the lungs. Unfortunately, blood could not be removed quickly enough to reduce the overload, and the infant died in a few minutes. At autopsy acute pulmonary œdema was found.

Exsanguination.

The earlier articles (Wiener and Wexler, 1951; Diamond, Allen and Thomas, 1951; Walker and Neligan, 1955) stressed the removal of more blood than was injected, which is extremely important in a certain type of case mentioned earlier. In more recent articles, the importance of not having too large a negative balance in an infant not severely anæmic is stressed (Walker and Mollinson, 1957; Allen and Diamond, 1958). Exsanguination is likely to occur in an infant who is deeply jaundiced, but whose hæmoglobin level is high, who is quite well and who has no circulatory overload. Three of the unexplained deaths in this series were, I believe due to this.

Earlier in the series I used always to build up a negative balance of 40 to 60 ml. by removing 20 ml. and injecting 10 ml. Three infants later in the exchange became intensely restless and irritable, and developed a pulse rate between 180 and 200 beats per minute. The infants' blood while being withdrawn was noticeably darker. After about 20 minutes like this they suddenly became pale and blue, limp and cold, with rapid sighing respirations and a slow, weak pulse. The venous pressure was estimated, but the column of blood disappeared rapidly into the umbilical vein. Despite all efforts and abandonment of the procedure, the infants died within two hours. On the next occasion when this happened, I tried the effect of injecting 30 ml. of blood and found that the infant's condition returned to normal in about half an hour.

Circulatory Stress.

The rather vague term circulatory stress used by Farquhar and Smith (1958) describes those infants who exhibit signs due to gross changes in the circulation. These authors described a picture of pallor of the upper half of the body and cyanosis of the lower, and they wondered whether it was due to opening of the ductus arteriosus, producing a veno-arterial shunt. I have seen two infants exhibit mild generalized cyanosis, with large irregular-shaped blue patches on the face, arms and trunk, which lasted for several hours. It was thought that this was due to a spasm of the great veins producing severe venous stasis in localized areas. Another infant, soon after the conclusion of an exchange, developed marked cyanosis, and was found to have a widespread loud systolic murmur, gross cardiac enlargement and electrocardiographic changes suggestive of a transposition of the great vessels. A week later no abnormality could be found.

Cardiac Arrhythmias.

It is possible that the stress of exchange transfusion may produce serious cardiac arrhythmias, such as ventricular fibrillation. A continuous electrocardiographic examination was performed during three exchanges. The infant during one of these developed shock, but no change was found in the record, apart from sinus tachycardia.

Autonomic Stress.

This also rather vague term used by Farquhar and Smith (1958), describes the changes perhaps due to stimulation of the umbilical vein, ductus venosus, portal vein or inferior vena cava by the catheter or injection or withdrawal of blood. This, in my view, is extremely important. The infants in whom this is likely to occur exhibit constant restlessness and irritability and cry loudly when blood is being removed or injected. Their pulse rate is 140 per minute or more, and they tend to vomit clear mucus. Blood is difficult to withdraw,

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

and frequent movements of the tip of the catheter are necessary. Quite suddenly the withdrawn blood will become dark, and if the exchange is not stopped and the infant rested, the clinical picture of shock with pallor, coldness, cyanosis, flaccidity and absence of a column of blood in the catheter will appear. Such changes also may appear if the exchange is being carried out too rapidly or the blood injected too quickly, causing sudden distension of the portal veins. They also seem to be more likely in infants of lighter birth weight, especially if the exchange is being carried out several days after birth, when functional closure of the ductus venosus has already proceeded to some extent. It is believed that irreversible shock from this cause was responsible for the deaths of the remaining six babies whose fatal collapse could not be explained at autopsy.

Recommendations.

The foregoing brief review of the possible causes of collapse during exchange transfusion embodies my observations and the scanty literature on the subject. Certain rules have been developed which have resulted in no deaths in the last 160 exchange transfusions. The recommendations which follow are believed to be essential to avoid danger in the procedure.

The blood to be used should have been collected within three days of its use. For anæmic infants the blood should not be shaken, and the cells should be allowed to remain settled after being removed from storage. If the blood is fresh and the packed cells from this bottle only are used, excess potassium in the blood injected into the baby will be at a minimum.

Immediately the blood has been cross-matched it is placed in a basin of water maintained at 100° F. This blood is not injected into the infant until it is judged to have reached this temperature. This takes at least half an hour. During the exchange the blood is kept at this temperature, it being a nurse's responsibility to watch the thermometer in the water surrounding the blood and to add hot water if necessary. The apparatus used is illustrated (Figure I). The danger of using

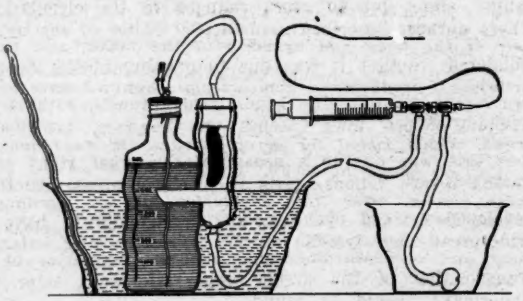

FIGURE I.

blood much colder than the body temperature of the infant is thus largely avoided. A better arrangement is for the transfusion tubing to be coiled in a water bath kept at a constant temperature of 100° F. (Marting, Wagoner and Wilson, 1955), but such apparatus is expensive.

The catheter used must be soft and the K.31 infant feeding tubes manufactured by Pharmseal Laboratories, Glendale, California, are ideal.[1] The operator must be gentle in inserting the tube. Rough handling is likely to cause venous spasm and subsequent difficulty. The tube will always go in with patience, although on rare occasions it may take 20 minutes. Once the tip has passed the obstruction usually encountered at the umbilicus, the catheter slips in easily. It should be advanced until blood flows into the lumen and then about 1 cm. farther. Inserting the catheter too far causes undesirable pressure changes in the circulation, leading to circulatory stress. These catheters have no marks, so it is wise to have a duplicate or a piece of thread the same length so that one can ascertain how far in is the tip. It should not be inserted more than 8 cm. from the abdominal wall.

[1] Available from Drug Houses of Australia.

It has been advised (Diamond, Allen and Thomas, 1951) that the venous pressure should be reduced by removing 20 ml. of blood and injecting 10 ml. till the column of blood in the catheter is 6 cm. vertically above the umbilicus. This is thought to be too low, as most infants in good condition have a venous pressure between 4 and 10 cm. It probably does not become dangerous until it exceeds 15 cm. This measurement of venous pressure can be most inaccurate, for any of the following reasons: the infant may be restless; a small vein may be tightly gripping the catheter; or a small clot may form in the taps and be forced down, partly blocking in the holes at the end. It is preferred to estimate on the condition of the infant the negative balance to be used. Very sick, partly hydropic infants with cord hæmoglobin levels below 8 grammes per 100 ml. should be given an exchange with a negative balance of 50 to 60 ml.; infants not hydropic, with a cord hæmoglobin value between 8 and 12 grammes per 100 ml., 30 to 40 ml.; and infants with a cord hæmoglobin level above 12 grammes per 100 ml. as well as all infants in good condition after the first day, 10 to 20 ml.

It is not impossible to canulate one of the umbilical arteries, with the result that the catheter rapidly fills with bright red blood, which spurts in jets out of the end. This is an easy and rapid way of giving the infant an exchange transfusion; but of course one must not try to reduce this arterial pressure, a mistake which has occurred.

It should be one person's sole duty to record the blood removed and injected. The operator should say how much has been removed after the blood has been injected into the waste bowl, and how much has been injected after it has actually entered the infant. He should make certain that he has been heard by the recorder before proceeding further. Concentration flags in a long exchange, and it is easy to inject or remove twice in succession; this may lead to overloading or exsanguination.

Estimation of the venous pressure is carried out after injection of each 100 ml.; but, as was mentioned earlier, this can be inaccurate. The ideal infant is asleep throughout, and it has been invariably found that a sleeping infant is in no danger of collapse. Conversely, a crying, struggling infant should be carefully watched. The catheter in use should lie on a white swab, and the colour of the withdrawn blood should be watched constantly. If it is noticeably paler than the blood being injected, the infant's circulation is satisfactory. If it should become dark, it is dangerous to continue.

The extraction and injection of blood should be done without force. The plunger of the barrel must not be sticky, and the blood must be extracted by a gentle rotatory movement of the plunger. Too vigorous extraction causes spasm of the vein, and no amount of force will then get blood. If no blood can be removed, the tip of the catheter should be advanced about half a centimetre and extraction tried again. If no blood comes, this manœuvre should be repeated. If it is still impossible to get blood, the operator should look for clots in the lumen of the taps or in the proximal end of the catheter. If blood still will not come, the catheter should be removed, and usually a clot is found blocking the apertures at the tip. A fresh catheter can be easily inserted. Too frequent moving of the tip of the catheter irritates the vein and causes spasm. Injection of blood should be gentle. If it is injected too fast, the infant will squirm and even cry violently, and if this stimulus occurs too much, autonomic stress from distension of the vein occurs, with possible collapse.

The speed at which an exchange is conducted is of the greatest importance. With good apparatus, a good catheter and injecting and removing 20 ml. aliquots, it is possible to exchange one litre of blood in one to one and a half hours. In my opinion this speed is extremely dangerous, and such an operation will inevitably cause collapse and possible death of an infant eventually. Aliquots extracted and injected should be 10 ml. and large amounts should not be used, although such large amounts will reduce the total time of the exchange considerably. It is a good rule to take 15 minutes for the exchange of 100 ml. It is the recorder's responsibility to keep the time for each 100 ml. exchanged, preferably with an X-ray timer, and if a shorter time than 15 minutes has been taken, the operator should leave transfusion blood in his catheter and taps and wait till 15 minutes has been reached before proceeding further. Should the blood being withdrawn from the infant become darker, usually a rest of five to ten minutes will make the blood go pink again. If one proceeds at this pace, the average exchange of 800 ml. will take about two hours, which is quite fast enough. The theoretical greater efficiency of exchanging in 20 ml. rather than 10 ml. aliquots is too small to be of any significance.

Calcium gluconate solution (10%) in amounts of 1 ml. is given after each 100 ml. aliquot of blood has been injected. I have carried out 44 exchanges without using it, and have never seen tetany appear. It does cause bradycardia while being injected, but probably does no harm. Whether it is beneficial is doubtful.

Should an infant develop cyanosis, pallor, coldness and flaccidity, and should the extracted blood grow darker, the exchange must be stopped and the venous pressure measured. If it is high—over 15 cm.—30 ml. of blood should be extracted, the venous pressure being measured after each 10 ml. aliquot is removed. Should there be signs of acute pulmonary œdema, 50 ml. should be removed in this way. The catheter should then be filled with isotonic saline and the exchange suspended for half an hour. If the infant is not back to normal in this time, the exchange should be terminated and resumed after the infant has recovered—about six hours later. If the venous pressure is low and the column of blood absent, 30 ml. should be injected in 10 ml. amounts, and the infant rested for half an hour. If he has not recovered in this time, the exchange should be suspended for some hours.

Conclusion.

The observations and recommendations given in this paper are the result of personal experience. Probably laboratory and other investigations will prove the observations fallacious and the recommendations wrong. Exchange transfusion for jaundice in the new-born is a very valuable form of treatment, and will be carried out far more frequently than it now is, and rightly so. However, it is no procedure for the inexperienced, and it is hoped that this paper will help those who, owing to unavoidable circumstances, have to treat such infants by exchange transfusion without a background of training and practice.

Summary.

The possible causes of collapse during exchange transfusion in the new-born are discussed. Precautions to avoid such collapse are detailed.

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STAPHYLOCOCCAL EMPYEMA.[1]

By E. S. STUCKEY,
Sydney.

ONE of the most fascinating aspects of medicine and surgery is the changing pattern of disease processes. With the advent of antibiotic therapy we came to regard pneumonia as a disease no longer likely to lead to surgical complications, and even when empyema occurred we thought that it should clear up with simple aspiration. This is no longer true, as you all know.

During the winter of 1955, I was asked to operate on half a dozen patients with empyema, in nearly all cases due to staphylococci. Since then we have all become familiar with empyema as a frequent and dangerous complication of staphylococcal pneumonia and septicæmia. The organism is usually resistant to penicillin and often also resistant to the tetracycline group of antibiotics. This problem is not peculiar to Australia, as a survey of recent literature rapidly reveals. In April, 1955, at the annual meeting of the American Association for Thoracic Surgery at Atlantic City, New Jersey, W. E. Bloomer and others, of New Haven, Connecticut, presented a paper on 11 cases of staphylococcal empyema in infancy encountered in ten years. During the ensuing discussion, John Snyder, of Bethlehem, Pennsylvania, quoted seven cases encountered in one winter, with three deaths; G. M. Brownrigg, of St. John, Newfoundland, spoke of 16 cases in five months, with six deaths; E. D. Gagnon, of Montreal, reported a series of 47 cases in six years; and D. Miller, Burlington, Vermont, had encountered 25 cases in five years. Mackenzie and McKim (1956), of London, Ontario, encountered 10 cases in four months with no deaths. Nearly all these patients were infants.

In November, 1957, Fisher and Swenson, of Boston, reported the results of treatment of 18 infants, and gave an informative summary of the pathology and treatment of the disease. Charles V. Pryles (1958), also of Boston, described 24 cases of staphylococcal pneumonia in infancy in 19 of which either simple empyema or pyopneumothroax developed. There were nine deaths. James Baird of Wellington, New Zealand, has recently (1958) reported 32 cases of empyema in five years, some being in adults.

I have extracted the admissions to the Royal Alexandra Hospital for Children, Sydney, in the three-year period from 1956 to December, 1958, and find that we have had 68 cases of empyema complicating pneumonia in that period, with 13 deaths; nearly all were due to staphylococci. Of these patients, 42 were aged 12 months or less, and all 13 deaths were in this age group. It is obvious, therefore, that this is a serious problem, which concerns both physician and surgeon. I have not included empyema occurring as a complication of thoracic surgery; nor have I included cases of pneumonia with non-suppurative effusion.

It is worthwhile, even in this gathering, to summarize the pathological features of the disease process. The pneumonic infection is almost certainly staphylococcal from its onset; but it frequently occurs during a relatively innocent attack of upper respiratory infection or

[1] Read at a meeting of the Australian Pædiatric Association, Canberra, April 17 to 20, 1959.

after an illness such as gastro-enteritis. Not uncommonly it occurs as fulminating staphylococcal septicæmia without warning and with overwhelming toxæmia. Nearly all our deaths were of this nature. All radiological, surgical and autopsy evidence agrees that in the early stages there is patchy consolidation in one or more lobes, with rapid coalescence of the areas of consolidation. There is early seropurulent effusion, and in a surprisingly short time there are multiple peripheral intrapulmonary abscesses of varying size, some of which rupture into the pleural cavity, and many of which have eroded the wall of a small bronchus. It is not surprising, therefore, that the empyema which so frequently ensues is usually a pyopneumothorax with a broncho-pleural fistula, and that it frequently involves the whole hemithorax, with collapse and compression of the non-consolidated areas in the affected lung. Fibrin formation is marked, on the parietal wall as well as on the diaphragm, the mediastinum, and the collapsed lung, with a marked tendency to loculation and to persistence of the cavity if it is not treated adequately.

During recovery there are frequently large and small peripheral air cysts in the lung parenchyma; these are usually thin-walled, but sometimes thick-walled abscess cavities with fluid levels. Fortunately both intrapulmonary air cysts and extrapulmonary fibrosis have a marked ability to clear up, if the empyema has been adequately drained, and the organisms have been killed by the body defences and by suitable antibiotics. Some patients, however, do not improve until the chronic empyema cavity is excised, with or without removal of lung tissue which is the site of persistent bronchopleural fistula or chronic lung abscess.

All authorities are agreed that prompt and energetic treatment of staphylococcal pneumonia is necessary if life is to be saved. Such treatment includes the use of at least two antibiotics ("Chloromycetin" and erythromycin is a favourite combination, pending isolation of the organism and sensitivity tests). Other essentials in medical management are adequate oxygen, cardiac stimulants for gravely ill patients and transfusions whenever the hæmoglobin value falls. Early aspiration at the first sign of fluid is generally agreed upon, if only for diagnostic purposes and isolation of the organism. Despite early and adequate treatment, however, empyema frequently develops.

There is a tendency to rely on repeated needle aspirations in managing empyema. I wish to make a plea for early surgical consultation in all such cases. The overseas authorities whom I have already quoted are remarkably unanimous in their emphasis on early and adequate continuous drainage of the empyema cavity, whether or not there is air as well as pus present. I should like to quote some of these authorities. G. B. Forbes and G. L. Emerson (1957) from the University of Rochester, New York, state that: "Empyema has been treated by aspiration and instillation of antibiotics with varying success. The usual experience, like ours, is that any appreciable collection of pus is more effectively treated by drainage." They consider the early intercostal insertion of a catheter essential and even urgent, and advise constant suction, especially when a bronchopleural fistula is present. Early decortication is not advised; but in late cases with crippling fibrothorax they recommend radical surgery. Sound surgical principles have not been displaced by antibiotics, and they state that "the surgical treatment of the pleural complications of staphylococcal pneumonia is an emergency procedure".

Gabriel Seley, of New York, uses closed drainage through a wide-bore rubber tube early and freely. He does not use aspiration at all as a treatment, because relief is minimal and transient, and the needle may injure the lung. MacKenzie and McKim, of Ontario, Canada (1956), recommend early intercostal drainage with suction as soon as the diagnosis is established. Drugs may be instilled daily, or enzymes such as varidase if there is a tendency to loculation. They state: "We are quite convinced that drainage and complete evacuation of the pleural space is of foremost importance." They found no need for decortication in their 10 cases. Pryles, of Boston (1958), recommends early drainage, but stresses that it must be effective. He recommends instillation of bacitracin (10,000 units) at the first diagnostic aspiration. Swenson and Fisher (1957) also recommend long-continued closed intercostal catheter drainage. Early decortication is not advised; but some patients grow progressively worse, and need late resection of the exudate and affected lobe.

If I may summarize, my present views are that from the onset of empyema, with or without pneumothorax, a surgeon should take part in the team management, and that intercostal catheter drainage, with underwater seal, or with suction, is essential. Instillation of antibiotics or of fibrolytic enzymes may be of value early in the disease. At the first sign that the catheter is failing to keep the cavity drained, whether from loculation or from fibrin blockage. I believe rib resection with breaking down of loculi and the insertion of a wide-bore tube instead of a catheter should be undertaken. If, despite these measures, the lung fails to expand within a few weeks, more radical surgery should not be delayed; but the presence of thickened pleura or of pneumatoceles is not in itself an indication for radical surgery.

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Reports of Cases.

MITRAL VALVOTOMY IN A SEVEN YEAR OLD GIRL.[1]

By DOUGLAS STUCKEY,

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THE necessity for the surgical relief of mitral valve obstruction of rheumatic origin must arise very rarely in the first decade of life, and reports of only three such cases have been found in literature. The youngest patient was aged four and a half years at the time of operation (Bailey and Bolton, 1956), and the others were a seven year old Bantu boy (Bradlow and Crawshaw, 1955) and an eight year old Italian child (Angelino *et alii*, 1956).

Clinical Record.

The present patient was a girl, aged five years when she was first referred by Dr. Robert Vines in November, 1955. She had been suffering from recurrent cough and fever for five months. A cardiac murmur had been noted for the first time during this illness, and she had been treated as suffering from juvenile rheumatism. The sedimentation rate was elevated on only one occasion.

On examination of the patient, there were no clinical signs of rheumatic activity. The cardiac impulse was left ventricular in type, and there was a loud systolic murmur with thrill maximal in the fourth left intercostal space. There were no physical signs of pulmonary hyper-

[1] Read at a meeting of the Australian Paediatric Association, Canberra, April 17 to 20, 1959.

tension, and no diastolic murmurs were heard. An electrocardiogram was normal, as were the X-ray appearances of the heart and lungs, except that the main pulmonary artery and its branches were a little more prominent than usual. The evidence for rheumatism was felt to be unconvincing, and a provisional diagnosis of ventricular septal defect was made.

The child was examined again in March, 1958, when she was aged seven years. Apart from frequent colds and bronchitis she had remained in reasonable health until a few months previously, when she had become progressively shorter of breath on exertion, and was unable to keep up with other children running, playing, even walking any distance. The physical signs had changed dramatically, and were now characteristic of tight mitral stenosis complicated by pulmonary hypertension. An electrocardiogram showed early P mitrale and some evidence of hypertrophy of both ventricles, the right predominating. On X-ray examination there was slight generalized cardiac enlargement, but the main pulmonary artery and its proximal branches were grossly dilated. Only borderline enlargement of the left atrium could be demonstrated on X-ray examination with a barium bolus. At cardiac catheterization there was no evidence of left-to-right shunt, blocked pulmonary capillary pressures were greatly elevated, and very severe pulmonary hypertension was demonstrated, with pressures in the pulmonary artery substantially higher than systemic pressures.

At operation in May, 1958, by Dr. Douglas Cohen, tight mitral stenosis was found, the orifice admitting only the tip of the little finger. The valve cusps were pliable and only lightly gummed together at the commissures. These split easily with finger pressure out to the valve ring in both medial and lateral directions. The post-operative course was uneventful, no mitral murmurs being audible until the twelfth day, when a soft, short, mitral diastolic murmur could again be heard.

Since operation the child has remained reasonably well; but a thrill and loud systolic murmur in the fourth left intercostal space have again become evident. The possibility of ventricular septal defect as an underlying lesion has been considered, and it is proposed to explore this possibility by a second catheterization in the near future.

Summary.

A girl, aged seven years, has been submitted to mitral valvotomy for the relief of severe mitral valve obstruction. Tight mitral stenosis complicated by severe pulmonary hypertension developed under observation in less than two and a half years.

Reviews.

Physiotherapy in Obstetrics and Gynæcology (Including Education for Childbirth). By Helen Heardman; revised by Maria Ebner, M.C.S.P., with a foreword by W. C. W. Nixon, M.D., F.R.C.S., F.R.C.O.G., and Veronica Shand, S.R.N., S.C.M., M.T.D.; Second Edition; 1959. Edinburgh and London: E. and S. Livingstone, Limited. 8¼" x 5¼", pp. 256, with 97 illustrations. Price: 20s. (English).

THE untimely death of Helen Heardman has deprived physiotherapy and allied professions of an experienced worker, especially in the field of obstetrics and gynæcology. It is, therefore, encouraging that Maria Ebner has found time to produce a new addition of "Physiotherapy in Obstetrics and Gynæcology".

Few additions and alterations have been made to the original production. There is a description of the anatomy and physiology of reproduction, which, although superficial, is quite adequate for the purpose. The chapter on drugs has been revised, and includes the use of the antagonists to the morphine-pethidine group of drugs. In the section on anæsthesia no mention is made of the use of "pudendal block", which has many advocates today.

Throughout the book emphasis is placed on physical and mental preparation for childbirth. "The experience of childbirth—in triple, physical, mental and spiritual perfection, makes further contribution to the richness of family life." Statistics on a series of 800 labours from Leeds Maternity Hospital and University College Hospital have been included. The author is well aware of the difficulty of assessing the results of such training on a statistical basis. This book is worthy of consideration by midwives and obstetricians as well as by physiotherapists, for whom it was primarily written.

The Healing Voice: Treatment by Hypnosis. By A. Philip Magonet; 1959. London, Melbourne, Toronto: William Heinemann Ltd. 7¾" x 5", pp. 216. Price: 22s 6d.

THIS book is apparently intended for the layman, rather than for the medical practitioner. Anyone who contemplated taking a professional interest in medical hypnosis would surely demand something more sophisticated.

A short account of the history of hypnosis is followed by chapter 2, "What is Hypnosis?". The first sentence reads thus: "Hypnosis may be said to be a temporary state of mind, induced in a subject by the suggestions of a hypnotist—a state in which any further suggestions given by the hypnotist are acted upon by the subject implicitly." This is the type of statement which those of us who are interested in the history of hypnosis read in the works of the magnetizers of 100 years ago. There is now abundant proof that even the deeply hypnotized patient never reaches the state in which suggestions of the hypnotist are acted upon implicitly. A few lines further on we read: "Hypnotism is absolutely safe". We believe that most thoughtful persons who have experience of hypnotherapy would regard this as a gross overstatement. On the next page occurs the statement: "You cannot be hypnotized if you do not wish to be". This is manifestly untrue. Many poorly integrated and suggestible persons can be hypnotized against their wish, and even in face of their active opposition to the hypnotic induction. Two lines further on we read: "While in a trance, you will not do or say anything which you would consider indecent or harmful". The whole experience of hypnoanalysis with the ventilation of repressed material surely gives the lie to this. And so on.

Much of the book is concerned with a layman's description of superficial psychotherapy, and there are innumerable short case histories of successful treatment by hypnosis.

This is the age in which it is quite the custom to present technical medical matters to the lay public; but surely this should only be done accurately, and in such a way as to give some balanced view of the over-all picture. In the past, both the lay public and the medical profession have been led astray by inaccurate and over-enthusiastic accounts of hypnosis. Strangely enough, this book is published at the very time when a serious-minded group of workers of many countries has formed an international society which aims to promote the accurate and scientific investigation of hypnosis.

Urology in Outline. By T. L. Chapman, Ch.M., F.R.C.S. (Eng.), F.R.F.P.S. (Glas.); 1959. Edinburgh and London: E. and S. Livingstone, Limited. 8¾" x 6", pp. 184, with 138 illustrations. Price: 27s. 6d. (English).

THE importance of teaching urology to students is emphasized by the appearance of a further book devoted to the fundamentals of the specialty.

The presentation is unorthodox, in that each chapter consists of two or three pages of text followed by numerous black and white diagrams. The author rightly states that most people carry information in their minds as successions of simple mental images, and it is on this principle that he has produced this book. The work is purely an outline, and there is not enough detail to satisfy the average student; but there is no doubt he could gain a lot from it if he absorbed the points that are emphasized.

The chapter on benign prostatic enlargement is good, and the text gives a clearer description of the clinical course than most larger text-books. The author relies on an excretion urogram to assess the amount of residual urine, but we think that in hospital there is little risk in passing a catheter, which can be left *in situ*. Excretion urography is expensive and often time-consuming when there is poor renal function. One wonders why Freyer's operation is mentioned except as an historical note, and Harris's permanent cystotomy tube is evidently not known.

Although it is stated that the great majority of calculi cast a shadow on the X-ray film, the student is left without information about the radiotranslucent uric acid stones.

The chapter on hydronephrosis will be helpful to the student; but surely it would have been better to portray the modern Scardino-Culp operation rather than von Lichtenberg's, which the author himself states carries a risk of stenosis.

On the whole, however, there is little to criticize in the subject matter, except that there could be a little more detail in the text. It is a very easy book to read, succinct, up to date and quite cheap, and it does fulfil the author's hope that it "will be helpful to those who would like to acquire quickly an elementary knowledge of this subject".

Chest and Heart Disease in the Commonwealth. 1959. London: National Association for the Prevention of Tuberculosis. 8¼" x 5½", pp. 443, with illustrations. Price not stated.

THIS volume contains a full report of the many important discussions which took place at the NAPT Commonwealth Chest Conference held in London in July, 1958. The National Association for the Prevention of Tuberculosis (NAPT), as its name implies, was originally founded to combat tuberculosis, but has latterly extended its activities to cover all chest conditions, and has recently changed its name to The Chest and Heart Association. Though one of the twenty sessions was devoted to discussing the heart patient in everyday life, the 1958 conference was still chiefly concerned with pulmonary conditions, with the main emphasis on tuberculosis. It is necessary to mention only a few of the more important sessions to indicate the range of subjects discussed. A discussion on the world anti-tuberculosis campaign was opened by Sir Harry Wunderly, and provided a most interesting picture of the degree of success already attained together with an idea of the immensity of the problem still remaining. The session entitled "The Family and the Patient with Chest Disease" illustrates the social aspects of chest diseases, which formed an important part of the conference's discussions. In the session devoted to lung cancer, contributions were made by consultants in medicine, surgery and radiotherapy, by an almoner (presenting the patient's point of view), by a general practitioner, by research workers, by a clergyman (himself a cancer sufferer), by a psychologist and by a medical officer of health. One of the more specialized sessions was that on fungous disease, pneumonoconiosis and sarcoidosis. Finally, the session on BCG vaccination provided some interesting information about the present status of this procedure in Britain, where it has been simplified by the increasing popularity of the Heaf multiple-puncture apparatus, and by the elimination of conversion testing; since 1958 the British Ministry of Health has offered BCG vaccination to all thirteen-year-old schoolchildren. More than most conference reports, this unpretentious volume offers something of interest on a wide variety of topics.

Problems of Addiction and Habituation. Edited by Paul H. Hoch, M.D., and Joseph Zubin, Ph.D.; 1958. New York, London: Grune and Stratton, Incorporated. 8½" x 5½", pp. 264, with illustrations. Price: $6.50.

THE fifteen papers in this volume, which make up the proceedings of the forty-seventh annual meeting of the American Psychopathological Association, held in February, 1957, cover many facets of the problems of addiction and habituation. They include one paper on pica in children as an early pattern of addiction, and one on the physiological and psychological effects of the use of coffee. The presidential address by Howard Liddell of Cornell University is entitled "A Biological Basis for Psychotherapy". The paper of the most general interest, in that it is not primarily a medical discussion at all, is one on the documentation of the narcotic addiction problem in the United States by W. Lee Speer. It presents a very interesting picture of the problem from the purely statistical and administrative view. We learn that in "the twenties" narcotic addicts in the United States were estimated to number a quarter of a million, but that a thorough combing of the country by all enforcement agencies in the mid-1950's found only 34,729 individuals who had used narcotics illegally during the four years of the search. Speer believes in the efficacy of Draconian legislation. In 1955 the State of Ohio enacted legislation imposing a minimum mandatory sentence of 20 years' imprisonment for convicted sellers of narcotics; during the next seven months 19 convictions were secured; according to Speers, the deterrent effect was such that the traffic dropped by 80%. In some other States relatively nominal sentences are still the rule, and these States are the main centres of the traffic. If these figures are substantiated, they are of interest to students of other aspects of law enforcement besides the drug traffic. The remainder of the contributions are written by psychiatrists for an audience of their fellows, and for the most part this is not a book to interest the general reader. The scope of the discussion is illustrated by some of the titles: "Withdrawal Convulsions and Withdrawal Psychoses", "Experimental Psychopharmacology and Behavioral Relativity", "Psychotherapeutic Management of Abrupt Drug Withdrawal", "Group Therapy for Chronic Alcoholics". The authors are mainly professors and teachers from American medical schools and teaching hospitals.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Peripheral Vascular Diseases: An Objective Approach", by Travis Winsor, M.D., F.A.C.P., with a foreword by Burrell O. Raulston, M.D.; 1959. Springfield, Illinois, U.S.A.: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications, Limited. 9" x 6", pp. 866, with 435 illustrations. Price: £6 5s. (English).

The author's aim has been to present "an objective approach to the understanding and treatment of patients with peripheral vascular disease" in the light of modern developments.

"Medicinal Chemistry: A Series of Reviews Prepared Under the Auspices of the Division of Medicinal Chemistry of the American Chemical Society", author: Wilbur J. Doran; edited by F. F. Blicke and R. H. Cox; associate editor: L. A. Woods; assistant editor: Harriet Geer; 1959. New York: John Wiley & Sons, Limited. London: Chapman & Hall, Limited. 9" x 5¾", pp. 344. Price: $12.00.

This volume deals with barbituric acid hypnotics.

"Metals and Engineering in Bone and Joint Surgery", by Charles Orville Bechtol, M.D., Albert Barnett Ferguson, Jr., M.D., and Patrick Gowans Laing, M.B., B.S., F.R.C.S.; 1959. Baltimore: The Williams & Wilkins Company. 10" x 6¼", pp. 194, with 119 illustration. Price: 88s.

Background material from metallurgy, corrosion engineering and the application of engineering principles to the locomotor system.

"A Manual of Bandaging, Strapping and Splinting", by Augustus Thorndike, M.D., F.A.C.S.; Third Edition; 1959. Philadelphia: Lea & Febiger. Sydney: Angus and Robertson, Limited. 7¼" x 5", pp. 156, with 125 illustrations. Price: 30s. 3d.

For "the inexperienced medical student, pupil nurse, or orderly".

"The Living Body: A Text in Human Physiology", by Charles Herbert Best, C.B.E., M.A., M.D., D.Sc. (Lond.), F.R.S., F.R.C.P. (Canada), and Norman Burke Taylor, V.D., M.D., F.R.S. (Canada), F.R.C.S. (Edin.), F.R.C.P. (Canada), M.R.C.S. (Eng.), L.R.C.P. (Lond.); Fourth Edition; 1959. London: Chapman and Hall, Limited. 9¼" x 6", pp. 768, with many illustrations. Price: 45s. (English).

A "junior" textbook with a new format in the present edition.

"An Atlas of Normal Radiographic Anatomy", by Isadore Meschan, M.A., M.D., with the assistance of R. M. F. Farrer-Maschan, M.B., B.S. (Melb.), M.D.; Second Edition; 1959. Philadelphia and London: W. B. Saunders Company. Sydney: W. Ramsay (Surgical) Limited. 10" x 6¼", pp. 759, with 1466 on 412 figures. Price: £8.

The first edition was published in 1951.

"Current Therapy—1959: Latest Approved Methods of Treatment for the Practising Physician", edited by Howard F. Conn, M.D.; consulting editors: George E. Burch, M. Edward Davis, Vincent J. Derbes, Garfield G. Duncan, Hugh J. Jewett, Clarence S. Livingood, Perrin H. Long, H. Houston Merrit, Walter L. Palmer, Hobart A. Reimann, Cyrus C. Sturgis and Robert H. Williams. Philadelphia and London: W. B. Saunders Company. Sydney: W. Ramsay (Surgical) Limited. 10½" x 7½", pp. 781. Price: £6.

The title is self-explanatory.

The Medical Journal of Australia

SATURDAY, NOVEMBER 28, 1959.

SAFETY IN THE HOME.

ACCIDENTS which might reasonably have been prevented are responsible for a great deal of suffering, disability and death, not least among children. F. W. Clements, in the first of three papers on accidental injuries in pre-school children, published in 1955,[1] stated that in Australia deaths from accidents were the principal cause of death of children from one to 14 years. The pre-school period was the most important, because of the high accident rates in children of those ages, and because they were the formative years during which a child's awareness of accidents and hazards should be developed. A great deal of investigation and thought has gone into the effort to appreciate the causes and to devise some means of control of accidents, particularly among children. Of work already done in Australia, attention may be drawn to the three papers by Clements and to the report in the issue of February 11, 1956, of a round-table conference on accidental poisoning in childhood, held at the annual meeting of the Australian Pædiatric Association in April, 1955, during which papers were presented by F. W. Clements, by Jean Allen and Howard Williams, and by John Beveridge.

One particularly disturbing fact which emerges from these and other surveys is that a very high proportion of accidents occur in what should be the sheltered atmosphere of the home. Pre-school children, of course, spend a large proportion of their time in the home, and have therefore a limited opportunity to be involved in accidents elsewhere. Acknowledgement of this fact, however, although it may put our statistical thinking in order, provides no warrant for complacency. The plain fact remains that far too many accidents happen to people of all ages in their own homes, and that the small child, with his limited capacity to accept any responsibility in the matter, is entitled to more safety in the home than he often gets. In most States of Australia, State Health Departments and other bodies concerned with health education paid particular attention in their last year's programme to the theme of prevention of accidents in the home, and some vigorous and imaginative campaigns were carried out, notably in Victoria, where a six-weeks home accidents survey in the Melbourne suburb of Malvern culminated at a meeting in the Malvern Town Hall, when the accident figures from the survey were disclosed and various modern ideas on health education were put forward. This home accident survey and similar surveys now being carried out in various parts of Australia owe much of their inspiration to the Corrigin project carried out in 1957 by the Western Australian Health Education Council. A full report of this project has just come to hand. Prepared by J. C. Carr, Executive Officer of the Health Education Council of Western Australia, it warrants our attention.

It is interesting to note that the Health Education Council of Western Australia came into being only in March, 1956, and that this particularly successful project was the outcome of its first major decision—namely, that the problem of home accidents should be given sustained attention for a minimum period of five years. A special committee was appointed by the council to investigate the problem, and to make recommendations for field work covering this five-year period. The Committee included the medical superintendents of the Princess Margaret Hospital for Children and the Fremantle Hospital, where most victims of serious home accidents are treated. It was early found that, although the mortality rates from home accidents were fairly easily computable, morbidity was almost impossible to assess, and there was a general lack of specific information on the subject in the State, particularly in relation to minor accidents. This was a serious handicap to the committee. A broad inquiry into the attitude in the community towards accidents in the home produced some interesting findings and impressions: (i) the public apparently regarded home accidents as either inevitable or the result of parental carelessness; (ii) there was an almost total unawareness that home accidents were a serious public health problem; (iii) the public regarded public health as concerned only with the investigation and control of disease problems and felt that home accidents were not properly a field for the activity of a public health authority; (iv) few parents were aware of the need for total protection of infants, with gradual safety education taking over as the child's mental and physical abilities increased; (v) given an understanding of the problem and the method by which they could help themselves, most parents appeared ready to act for themselves.

With the primary long-term objective of reducing morbidity and mortality caused by home accidents in Western Australia the committee set out to make the public aware that the problem existed, to get the public to do something about the problem for themselves, to teach parents the need for and value of protection and safety education of young children, and to increase the scope of safety teaching in the schools as the logical next step from home teaching. A second extremely important objective was to secure the acceptance of health education and of the Health Education Council by the community, with the long-range objective of covering the State with local volunteer citizen committees who would first have proved to themselves the value of self-help in health problems. As a starting point the Committee agreed upon the following principles: (i) the initial target group should be the parents of young

[1] MED. J. AUST., 1955, 1:348 (March 5), 388 (March 12), 421 (March 19).

children; (ii) legislation was of little or no value in tackling the problem; (iii) parents could not and should not be accused of carelessness; (iv) a method by which whole families were active participants within their home environments would be more likely to succeed than a massive assault using only mass communications media; (v) mass communications methods must be relied on to make the initial public impact. After investigation of home safety programmes used elsewhere, the American "notebook project" was selected as offering the best method of securing family cooperation in a community accident-prevention effort. An intensive Press and radio campaign was then launched with the sole aim of drawing public attention to the home accidents problem. It was realized that these media were of limited value in changing attitudes in a problem of this kind, but it was hoped that the publicity, combined with various other approaches to community groups, would stimulate one or more local areas to action. No mention was made of the notebook project during the campaign. Both Press and broadcasting organizations cooperated fully.

Some eight months later the Mothers' Discussion Group in the country town of Corrigin, 160 miles inland from Perth, wrote to the Health Education Council asking for help in running a small home safety campaign in their district. In this action the group had been influenced by radio broadcasts on home safety. The Health Education Council's executive officer immediately visited Corrigin to suggest the notebook project to the mothers' group. They quickly saw that it was too big for them to organize alone, and agreed that other community groups should be drawn in. An impromptu meeting of representatives of the larger community groups with some individual citizens took place, and it was immediately evident that a strong community spirit existed in the district. This new group expressed the unanimous wish to organize the project for themselves, provided a public meeting was held to put the home accidents problem and the notebook scheme before the people of the district. The group acted as a steering committee to organize the public meeting and to arrange suitable publicity. The public meeting, which was attended by about two hundred people, voted to go ahead with the project, and an organization was formed, drawn from various community organizations together with a number of private individuals. It was known as the Corrigin Project Committee. After a full discussion of ways and means and details, notebooks were distributed to a little more than half the families in the district. The essential feature of these notebooks was a series of ruled columns providing space for the detailed recording of every accident, minor or major, occurring in the home.

The project began on June 17, 1957, and continued until October 7, 1957. For various reasons not all the notebooks were completed, but finally 132 complete notebooks were available for analysis. The committee of group leaders met weekly, and despite occasional difficulties enthusiasm for the project remained high throughout. The first and most obvious result was a substantial reduction in home accidents during the 16-week period. From the initial rate of 5·0 accidents per person per year in the first week, the rate dropped to 0·9 per person in the last week of the survey. Accidental poisonings had been a particular problem in the district, and it is notable that the number of these was exceedingly few during the survey. The issue of a pamphlet dealing with accident situations is thought to have prompted many people to eliminate a good deal of accident potential before the survey commenced. The local school cooperated in full, and not only encouraged the survey but carried out one within its own environs. The report states that as the project developed it became a matter of social pride among the children to have no home accidents to report.

Discussing subsequent developments the report points out that the ultimate test of any health education programme is "the extent to which people have acquired the desired information, have favourable attitudes, and are doing for themselves the things that improve their health". The Corrigin project is considered to have survived the test. From the beginning of the project in June, 1957, until the time of preparation of the report (September, 1958) there had not been a single case of accidental poisoning in the Corrigin district, and other forms of home accidents had been maintained at approximately half the pre-project rate. The committee of group leaders at the end of the project spontaneously decided to form themselves into a health education committee for the district, with the local medical officer of health as president, and it is intended to ask this committee to repeat this project after five years in order to determine the lasting effect of the notebook. The report points out that the Corrigin experience shows that once a community has demonstrated for itself, from its own experience within its own home and community environment, that self-help in health matters is not only possible but desirable and effective, the work of health education personnel becomes very much easier. This is vitally important in a State such as Western Australia with its great distances and limited resources. The home accident notebook project has therefore wider significance in Western Australia than the reduction of a major public health problem, although this in itself is the prime function of the project. Another significant post-Corrigin happening, which it is thought will accelerate development, is the acceptance by the Infant Health Service of the Department of Public Health of responsibility for stimulating notebook projects in the local areas in which they work. This is very important, as the Infant Health Service is established in the community and has the confidence of mothers. It is also encouraging to note that there is complete cooperation in this and other problems between the Department of Public Health, the Department of Child Health in the University of Western Australia, and the Health Education Council.

The report also gives a brief account of an unsuccessful notebook project undertaken by a Rotary Club in a suburban community. It is not necessary to go into details of this project here, but it underlines the

cardinal reasons given for the success of the Corrigin project: (i) The original request for help came from a single community group aware of the problem and wanting to do something about it. (ii) Enlistment of other community groups in a community programme was seen to be desirable if a sufficient number of families were to participate. (iii) The local people were made aware of the problem and decided to act together in solving it. (iv) Group leaders were elected by the people, with consequent acceptance both of leaders and of the programme. (v) The local people organized the project, made their own decisions on procedures, etc., and looked to their elected leaders for advice during the project. In summary, the report emphasizes, the Corrigin project demonstrated the effectiveness of community self help. In one sense at least, the Rotary Club project was not a failure, because it did teach certain important lessons. The most significant were, first, that patience is needed in health education work and, second, that people cannot and will not be pushed into adopting new attitudes and practices, unless they can be shown something worth while developing from their actions for themselves and for their children. Comparison of the Rotary project with the Corrigin project cleared the fog of theory behind which, to many workers, health education is obscured, so that perspectives for the future emerged clear and well defined. With general principles firmly established, work began in other centres which had already requested help in conducting their projects. In a series of districts the Corrigin pattern was followed closely with equal success, and another Rotary project, this time with a limited aim and careful planning, was also successful.

One of the most convincing features of this report is a copy of a letter written by the local medical officer of health nearly twelve months after the completion of the Corrigin project, which confirms the lasting effect of what had been done during the period of the project. The Western Australian Health Education Council is to be congratulated on this particular activity, not only because of the success of the Corrigin project itself and of subsequent similar and related projects in Western Australia, but also because of the stimulus that it has given to like action in other parts of Australia. It is to be hoped that, as experience grows of this method of promoting safety in the home, the results and the lessons learnt, together with any other ideas that are evolved, will be added to the common pool of public knowledge. Everyone agrees that adults should be encouraged to stop killing and maiming themselves and their children on the roads. It should be an even more obvious responsibility and a less difficult task to promote safety in the home.

Current Comment.

ELEVENTH ANNUAL REPORT OF C.S.I.R.O.

THE eleventh annual report of the Commonwealth Scientific and Industrial Research Organization, which covers the year ended June 30, 1959, is a long and technical document covering the wide varieties of scientific researches being undertaken by C.S.I.R.O. in such diverse fields as entomology, animal health, plant industry, forest products, industrial chemistry and radiophysics. It outlines work on hundreds of projects of importance to Australia's primary and secondary industries. Work on rain-making, mineral deficiencies of soils, sheep and wool research, prevention of evaporation and many other major projects are already well known. Other items of special interest have not received much publicity. It is to be noted, for example, that C.S.I.R.O. is to enter two new fields of research. Firstly, a wheat research unit has been formed, which will investigate factors affecting the quality of Australian wheat. Secondly, at the request of the Minister for Territories, C.S.I.R.O. has decided to establish a Rice Research Station on the coastal plains near Darwin. At this station studies will be made of nutrition and water requirements of rice and the possibility of developing varieties well suited to the local conditions.

Another item relates to the efforts of scientists at the Sheep Biology Laboratory at Prospect, New South Wales, to find out why newborn lambs die in bad weather conditions. The laboratory is equipped with special rooms in which cold and wet weather can be artificially produced. A striking difference in resistance to exposure has been found between lambs from well-fed ewes and lambs from poorly-fed ewes. It appears that even if the ewes have been well fed, it is imperative that lambs receive nourishment very soon after birth. In one interesting experiment maiden ewes (ewes which had not produced a lamb) and mature ewes were closely watched through pregnancy and lambing. It was discovered that over 20% of the maiden ewes abandoned their lambs, as compared with only 1% of the mature ewes. This seems to be a particular example of a very general observation, namely that animals (and birds) tending their first young are relatively inefficient when compared with more experienced breeders.

A BIOLOGICAL METHOD FOR THE CONTROL OF HOUSE FLIES.

BIOLOGICAL METHODS for the control of various unwanted organisms have been very useful, but one advocated recently in South Africa is quite unusual. J. J. Steyn[1] has described the use of the South African social spider (*Stegodyphus*) for the control of flies in kitchens, restaurants, dairies and anywhere else where flies congregate. The social spiders do not live separately, as do most other spiders, but dwell in large colonies in a large and very complicated nest made of cobweb. The spiders do not hunt for prey but remain in the nest and devour the flies which adhere to the sticky web. A community may consist of one hundred or more spiders, and the communal nests are sometimes very big—for instance, as big as a football. There are of course no spiders wandering round the walls and ceiling. These spiders with their nests have been used for a long time, until comparatively recently, by the Bantu tribes to keep flies and cockroaches out of their huts. The technique for using them is to hang a nest by a piece of string from the ceiling of the kitchen or other room, and that is all, except that if the flies on the web become too numerous for the spiders to handle, the excess may be wiped off with a broom.

Inquiry has shown that these social spiders are not found in Australia, so one has to depend on other means for keeping down flies. The various kinds of huntsman spider in Australia are very useful for getting rid of flies, moths etc., and are quite harmless to man. Unfortunately they have been mistakenly called tarantulas, and they do not find favour on the walls and ceiling with housewives, which is a pity.

[1] *S.A. Med. J.*, 1959, 33:730 (August 29).

Abstracts from Medical Literature.

PÆDIATRICS.

Spinal Cord Tumours in Children.

H. HAFT, J. RANSOHOFF AND S. CARTER (*Pediatrics*, June, 1959) state that in many children with spinal cord tumours at least a year elapses between the onset of symptoms and the diagnosis. This condition should be considered in those children who have disturbances of gait and complain of pain in the back or legs. Such a possibility is particularly strong if evidence of motor weakness in the lower extremities associated with abnormal reflexes and a sensory deficit is found on examination. Plain skiagrams of the spine and, when indicated, lumbar puncture and myelography, are necessary studies. The case histories of 30 children (15 years of age and under) with spinal cord tumours have been analysed by the authors. No significant age or sex distribution was found. A third of the tumours were intramedullary, and a half were extradural. Most of the tumours were located in the thoracic cord. A comparison of the types of intraspinal tumours found in adults and children reveals a greater incidence of meningiomas and neurofibromas in the adult group, while gliomas and congenital tumours tend to predominate in children. The aim of surgical treatment is to remove as much tumour as possible without producing irrevocable damage. Post-operative X-ray therapy was given to the spine in 20 cases and to lesions in the chest in four cases. Out of the whole series, 50% have done well; the prognosis is poor in those patients with neuroblastoma or sarcoma and in those developing an intracranial extension of the tumour.

The New York Programme for the Care of Premature Infants.

L. BAUMGARTNER *et alii* (*J. Pediat.*, June, 1959) have made a critical evaluation of the programme for the care of premature infants in New York City over the past decade. At the present time 11 approved premature centres are in operation, with a capacity of 327 beds. Statistics relating to mortality rates for premature infants in four weight groupings under 2500 grammes are presented for hospitals with approved premature centres and for the city as a whole. The survival rates in approved premature centres are generally higher than in hospitals not maintaining such centres. Although particular attention needs to be aimed at reducing the mortality rates in the category of those weighing 1500 grammes or less, the authors believe that much more emphasis than now prevails should be given to the care of infants in the categories of those from 1501 to 2500 grammes in weight, and consider that a significantly greater number of babies could be saved if "centre" standards of care could be given to all. The latter group not only comprises the majority of premature babies, but includes the ones which have the best potential for normal growth and development. In a discussion on the prevention of prematurity, the authors state that a higher proportion of mothers who were delivered prematurely had late or no pre-natal care than did those who were delivered at term. With the knowledge that the prematurely born infant is more vulnerable to environmental factors even after the neonatal period than is his full-term counterpart, the need for intensive follow-up care after discharge from the hospital is emphasized.

Cardio-Vascular Collapse due to Chloramphenicol.

J. M. SUTHERLAND (*A.M.A. J. Dis. Child.*, June, 1959) describes the death of three newborn infants occurring after large doses of chloramphenicol were administered intramuscularly to two infants and orally to a third infant. These three infants died with a clinical picture of vascular collapse. Two of the three were hypothermic. All three received doses of chloramphenicol significantly larger than are usually administered. There were no recognized pathological changes common to the three infants. The absence of post-mortem findings indicating any but minimal or subsiding infection in these three patients increases the likelihood that their deaths resulted from adverse drug action, although a clinical picture of vasomotor collapse and hypothermia may be associated terminally with overwhelming infections in the newborn infant. The fact that the three patients were all newborn infants raises the question of the possible peculiar response of the newborn to this drug. The author suggests after reviewing the literature that the adverse effect of doses of chloramphenicol of one and a half times the recommended amounts might result from any one or more of the following factors: rapid destruction of bacteria and release of endotoxin, drug hypersensitivity, methæmoglobin production, overgrowth of resistant organisms, toxic alternate metabolites, efficient absorption and/or poor elimination of drug, hypoglycæmia, disordered carbohydrate utilization, and adverse effect on the autonomic nervous system. He concludes that the report of these possible adverse results of chloramphenicol therapy may permit other more definite and better-controlled observation and indirectly result in a more rational application of a useful chemotherapeutic agent to the newborn infant with bacterial infection.

The Respiratory Distress Syndrome.

W. BAUMAN (*Pediatrics*, August, 1959) states that the respiratory distress syndrome of premature infants is often associated with death and pulmonary hyaline membrane formation. The newborn premature infant may manifest this syndrome by several physical signs. Characteristically, he breathes as if he had glottic obstruction; intercostal retractions accompany strong inspiratory movements. The flexible thoracic cage collapses as the abdomen distends under the strong contraction of the diaphragm. Hypoxia may be evidenced by cyanosis. Auscultation may reveal diminution of breath sounds and presence of rales. An expiratory grunt is frequently audible. Dilatation of the alæ nasi may be noted, and the chin occasionally descends and the mouth may open with inspiration. The respiratory rate is variable. During the first days of life the rate is usually rapid (over 60 per minute). Terminally it slows, with increasing periods of apnœa and eventual death. Œdema of the hands and feet is usually present. Affected premature infants develop the respiratory distress syndrome during the first 12 hours after birth. The intensity of dyspnœa increases until they are 36 to 48 hours old. Those who succumb usually do so at this time. Some babies who exhibit similar initial signs gradually show lessening of distress and slowly recover by the third or fourth day. Some children born of diabetic mothers also develop respiratory distress, as do occasional otherwise normal full-term babies. The crude fatality rate in the first seven days of life of dyspnœic infants in the author's study was 60% and, of those who died, half had hyaline membrane; whereas the fatality rate was 10% in those without distress and only one in ten of these subjects had pulmonary hyaline membrane. Since 40% of those who had clinical stigmata of the respiratory distress syndrome survived without treatment, any proposed therapy for this condition must be critically evaluated before it can be considered beneficial.

Insufflation of Air for Acute Intestinal Intussusception.

E. FIORITO AND L. CUESTAS (*Pediatrics*, August, 1959) call attention to a new method of diagnosis and non-surgical treatment of acute intestinal intussusception in infancy. It consists of insufflation of the colon with air under fluoroscopic and manometric control. By this technique full reduction was achieved in 94% of 86 cases of intussusception, only five patients requiring operation. The authors state that one advantage of this method is its simplicity, without the troubles and interruptions during the fluoroscopic examination commonly seen in the barium-enema technique. There is also fast diffusion of the air, so that it advances promptly to the intussusception, giving clear-cut visualization of the intussusceptum, which is seen surrounded by gas. They say there is no contraindication to the technique, which can be used in the diagnosis of advanced cases. As the air passes the ileo-cæcal valve in all cases, ileo-cæcal forms can therefore be diagnosed. The diagnostic stage is started by insufflating the colon slowly, working with an air pressure of not more than 60 mm. of mercury. An X-ray film is then obtained. In a few cases this pressure is enough to reduce the intussusception, but in most cases if there are no medical contraindications the air pressure is increased to 130 mm. of mercury. This pressure is maintained until the air completely fills the small bowel, which is checked radiologically, and thus full reduction of the intussusception is confirmed.

Miliary Tuberculosis.

E. LINCOLN AND F. HOULD (*New Eng. J. Med.*, July 16, 1959) report the results of various forms of antimicrobial therapy in 63 children with acute generalized miliary tuberculosis. Of a small group of nine who were treated with thiazol-

sulphone, six survived and remained well until two of them manifested renal tuberculosis and one pulmonary tuberculosis 11 or 12 years after the original diagnosis. The case fatality rate improved in the next group of 29 children treated with added streptomycin, but meningitis developed in six patients under treatment for miliary tuberculosis. In the third group of 25 children treated with the addition of isoniazid only one death occurred. The authors conclude that isoniazid is obviously the most efficient antimicrobial agent in the treatment of miliary tuberculosis. In this series fewer complications developed during treatment, and no child had meningitis while receiving isoniazid. The rate of clearing of the mottling in the chest skiagrams was slowest in the patients treated by thiazolsulphone only (32 to 44 weeks); in the two later groups, with added streptomycin in one group and with three antimicrobial agents including isoniazid in the final group, the average was 13 weeks. As better antimicrobial agents were used in the treatment, the amount of calcification seen both in the primary complex and in metastatic areas decreased. The authors conclude that observation of the extent and location of calcification in the primary and metastatic areas can possibly be utilized as a measure of the effectiveness of an antimicrobial agent.

Ulcerative Colitis in Childhood.

R. KING *et alii* (*Arch. Dis. Childh.*, June, 1959) report a follow-up study of 52 children with chronic ulcerative colitis, the period of follow-up ranging from one to 26 years, with an average of nine years. Eight had died as a result of the colitis, four had developed carcinoma of the colon, and 12 had undergone excisional surgery with one post-operative death. The over-all incidence of malignant degeneration was high, but even more striking was the early age at which the neoplasms occurred, the average age at the time of death from or diagnosis of the neoplasm being 25 years. There is now little doubt that chronic ulcerative colitis is a pre-malignant condition in both adults and children, the tendency for malignant change to occur increasing with the duration of the colitis. Four of the 24 patients who had had the disease longer than nine years developed carcinomas, a figure which the authors consider should weigh very heavily in favour of excisional surgery when the management of the long-standing chronic disease is being considered. The tendency now is to perform ileostomy with colectomy and rectal resection in one or two stages, although in some cases ileo-rectal anastomosis may be possible. The authors conclude by saying that a childhood condition in which 27% of the sufferers continue with chronic ill-health and from which 15% die, constitutes a serious challenge to medical science.

Scalp-Vein Intravenous Therapy.

Z. H. ZAIDI (*Canad. med. Ass. J.*, April 15, 1959) describes a protective device for the scalp-vein needle during intravenous therapy for infants. He points out that the needle is often displaced by the movements of the infant's head. The device is simple, consisting of an ordinary 1 oz. medicine cup made of stiff paper coated with wax. The bottom is trimmed to form a hinged lid, and a notch is made in the rim of the cup for the infusion tube. The cup is inverted over the needle and is attached to the infant's scalp by vertical strips of sticking plaster; the lid may then be opened to check the needle when desired.

SURGERY.

A Sarcoma Involving the Jaws in African Children.

D. BURKITT (*Brit. J. Surg.*, November, 1958) describes a sarcoma involving the jaws in African children which has become a distinctive clinical condition at Mulago Hospital in Uganda. It is the commonest malignant condition seen in childhood at that hospital. During the past seven years, 37 patients with this sarcoma in the jaws have been seen, and a further eight children were seen in whom the tumour was diagnosed clinically, but they have not been included in this series because of lack of histological confirmation. The patients were from two to 14 years of age and the tumour appeared to start in the region of the alveolar process of the maxilla or mandible. The teeth fell out, and the tumour grew rapidly, grossly distorting the face. Within two or three months of the onset of symptoms, the relatives removed the children from the hospital in a moribund condition. In 15 of the patients tumour deposits were demonstrated or strongly suspected in the adrenals, kidneys and liver. These were also seen in thyroid, testis, heart, stomach, pancreas, salivary gland, cranium or femur. Tumour deposits have not been demonstrated in lymph nodes or spleen. Radical surgery was done in only one case, and this was followed by recurrence. Radiotherapy has not been available. Histologically, it has not been possible to determine the exact nature of the tumour. The author considers that it is probable that the jaw tumour is a secondary deposit, possibly from an abdominal primary tumour. Neuroblastoma has been considered, but there is no histological evidence to support this diagnosis.

Intussusception.

R. STRANG (*Brit. J. Surg.*, March, 1959) presents a review of 400 consecutive patients with intussusception operated on at the Royal Hospital for Sick Children, Glasgow. Causative associated lesions were found in only eight cases; these were Meckel's diverticulum (five cases), adenoma of the small bowel (one case) and Henoch-Schönlein's purpura (two cases). In discussing the ætiology of the condition, the author observes (i) that the age group most commonly affected was that between the third and tenth months of age; (ii) that the series included 250 boys and 150 girls; (iii) that intussusception was preceded by enteritis in 18 cases; (iv) that in many cases the mesenteric lymph nodes were quite fleshy and inflamed. Out of 318 cases, in 89 (27%) there was a mild but definite degree of mesenteric lymphadenitis, in 16% there was a mesenteric lymphadenitis of moderate severity, and in 3·5% the condition was severe. The author states that it is reasonable to suppose that the condition of the mesenteric nodes reflects that of the lymphoid tissue in the wall of the intestine and that it has been suggested that mesenteric lymphadenitis may be of viral origin. Intussusceptions are more common in some years than in others, and at certain seasons of the year (winter and spring). The author suggests that it may be significant that viral infections are more prevalent in these seasons. In 53% of the entire series the intussusception was ileo-colic, in 34% it was ileo-caecal; there were 18 cases of the colo-colic type, and 25 cases of enteric intussusception. The average duration of symptoms before operation was 30 hours. In discussing treatment, the author considers the hydrostatic method of attempting reduction and summarizes the objections to its routine use; all patients in his own series were treated primarily by operation. In 33 cases the intussusception was irreducible; the author discusses the three methods which were employed in the treatment of irreducible intussusception. These were: (i) resection and primary anastomosis; (ii) simple anastomosis with return of the intussusception to the peritoneal cavity; (iii) exteriorization. The best results were obtained by exteriorization. There were 23 deaths in the entire series, of which 13 occurred in cases in which the intussusception was irreducible.

Acute Obstruction Caused by Hæmatoma of the Small Intestine.

A. L. MESTEL *et alii* (*A.M.A. Arch. Surg.*, January, 1959) state that they were able to find in the literature 19 cases of duodenal or jejunal hæmatoma due to blunt injury to the abdomen, and causing obstruction in children. They report two new cases. The most constant clinical findings were abdominal pain and vomiting and a raised leucocyte count. They state that X-ray examination confirmed the diagnosis. They consider that the condition can be treated with operation, by gastric suction and fluids, given intravenously. If surgery is performed because of misdiagnosis, then the hæmatoma should be evacuated without resection of the damaged bowel.

Shunt Procedures for Portal Hypertension.

C. E. SEDGWICK AND H. A. HUME (*A.M.A. Arch. Surg.*, March, 1959) report the results of clinical and laboratory follow-up studies in a series of 42 patients in whom direct venous shunts were carried out for portal hypertension. The follow-up periods varied from one to eight years. The overall late mortality rate was 45% and the operative mortality rate was 16%. They noted a decrease in the size of varices post-operatively in 12 out of 15 patients whose pre-operative and post-operative skiagrams were available for comparison. The authors state that factors related to failure of these procedures to afford even greater protection from hæmorrhage included the technical adequacy of the shunt, the status of the liver, and the subsequent development of complications such as subphrenic abscesses. They consider that there is little doubt that many of those who survived the operation would live for many years.

Medical Societies.

AUSTRALIAN PÆDIATRIC ASSOCIATION.

THE annual meeting of the Australian Pædiatric Association was held at Canberra on April 17 to 20, 1959, SIR KENNETH FRASER, the President, in the chair.

Election of Office-Bearers.

The following office-bearers were elected for the year *1959-1960*:

President: Dr. H. McLorinan.

Vice-President: Dr. Ralph Crisp.

Honorary Secretary: Dr. Norman Wettenhall.

Honorary Treasurer: Mr. Peter Jones.

State Representatives: Dr. R. Wall (Tasmania), Dr. Ivan Magarey (South Australia), Dr. D. G. R. Vickery (New South Wales), Sir Kenneth B. Fraser (Queensland).

Controlled Hypothermia in Childhood.

VICTOR HERCUS (Sydney) read a paper entitled "Hypothermia in Cardiac Surgery" (see page 785).

DOUGLAS COHEN (Sydney) read a paper entitled "Hypothermia for Cardiac Surgery in Children" (see page 786).

RUSSELL HOWARD (Melbourne) said that Melbourne had roughly the same procedure as Sydney, but arterialized blood was not used, and heparinized blood was not used. They had not had a case of ventricular fibrillation. He did not believe that hypothermia was necessary and thought it was in fact undesirable in coarctation of the aorta, as it was time-consuming and involved a much larger number of people. Fifty patients had been operated on without the use of hypothermia.

P. G. JONES (Melbourne) said he thought that hypothermia in a Blalock's operation might cause increased viscosity of the blood, with possible increase in thrombosis at the anastomosis.

PROFESSOR ORVAR SWENSON (Boston) said he thought that the use of hypothermia in open surgery was conditioned by the accuracy of the diagnosis. C. Walter Lillehei insisted that all cases could not be predicted, and that arrangements should be made for more extensive procedures than were allowed by hypothermia.

MARGARET MCCLELLAND (Melbourne) said she thought that the blanket designed by Dr. Moss Cass was simpler than that used by Dr. Cohen. She thought that hypothermia would be of great use in other circumstances, such as brain surgery for aneurysm or space-occupying lesions, in hyperpyrexia and in convulsions, and agreed that there was a real place for hypothermia in short cardiac operations such as those for atrial septal defect and pulmonary stenosis.

Dr. Cohen, in reply to Dr. Jones, said that they had found no cases of increased bleeding or thrombosis in hypothermia, and that clamping of one pulmonary artery of a very sick baby put a severe strain on the cerebrum, so relief of that strain by hypothermia helped such babies.

Oxygen Therapy in Pædiatrics.

R. GODFREY (Perth) read a paper entitled "Techniques for Oxygen Therapy in Infancy and Childhood" (see page 789).

J. H. COLEBATCH (Melbourne) asked if a box partly closed at the top, such as various types of premature baby bassinets, was more satisfactory.

Dr. Godfrey replied that such a box was satisfactory, provided draughts were excluded.

F. ARDEN (Brisbane) asked if there were any risks in giving too much oxygen.

Dr. Godfrey said he thought that the dangers of high oxygen concentrations had been exaggerated, except in relation to the newborn.

VICTOR HERCUS (Sydney) stressed the importance of considering adequate pulmonary ventilation as well as oxygen therapy.

T. G. MADDISON (Melbourne) asked if oxygen concentrations in "Isolettes" or "Insulcots" had been measured.

Dr. Godfrey replied that they had not.

Hæmatoma in the Posterior Fossa.

T. Y. NELSON (Sydney) read a paper entitled "Acute Subdural Hæmatoma in the Posterior Fossa" (see page 792).

M. T. COCKBURN (Adelaide) said that, from his experience, he could not agree that the presence of bleeding could always be recognized within 24 hours unless it was very severe.

Dr. Nelson, in reply, said that he did not believe it was necessary to recognize symptoms within the first 24 hours to diagnose birth trauma, and quoted Ingram's classification according to the month of life in which symptoms appeared; those in the first month of life could be accepted as due to birth trauma, and those in later months as due to other causes.

R. SOUTHBY (Melbourne) protested against the use of the nomenclature "birth trauma", especially when used by a consultant or in describing the condition to parents, as it implied that the trauma was caused by someone, mainly the obstetrician. He suggested as a substitute "hæmorrhage within the skull".

R. A. BARTER (Melbourne) mentioned that a woman in the German literature had stated the fact that meningoencephalitis due to Toxoplasma infection had been noted in the majority of cases of tentorial tear, indicating a high incidence of the infection in the mothers. She recommended that that cause should be recognized early and the mother treated, so as to obtain a healthy child in subsequent pregnancies.

Pulmonary Valve Stenosis.

MOSTYN POWELL (Melbourne) read a paper entitled "Pure Pulmonary Valve Stenosis" (see page 795).

RUSSELL HOWARD (Melbourne) showed a film of the most severe case of pulmonary stenosis in which operation had been performed at the Royal Children's Hospital to date. He said that the operation used was performed under hypothermia. A vertical incision was employed, the sternum being split from end to end. That provided a good exposure and avoided opening either pleural cavity. The pulmonary artery was then opened by a longitudinal incision, and the valve was split lengthwise along at least two lines of section. Most of the 17 patients operated on at the Royal Children's Hospital by that method were definitely improved. Two patients, without very high right ventricular pressures (60 to 80 mm. of mercury) appeared unlikely to show much improvement, though operation was too recent for certainty. One of the 17 patients died from an acute liver condition, which was shown at autopsy to have been superimposed on preexisting hepatitis. Most of the remaining patients had a comparatively benign convalescence, and all ultimately recovered to give a good result. Seven of the patients operated on were cyanosed before operation.

DOUGLAS STUCKEY (Sydney) commented that his experience in Sydney was very similar. The use of the pulmonary artery route under hypothermia was practised in Sydney. When doubt existed between the diagnosis of pure pulmonary stenosis and tetrad of Fallot, he sometimes used the manœuvre of injecting a drug such as "Methedrine" whilst the catheter was *in situ*. Such drugs produced a rise in systemic pressure without a corresponding effect on the right side of the heart unless a ventricular septal defect was also present.

DOUGLAS COHEN (Sydney) said that his experience was very comparable. He had started with the Brock closed technique, but after six cases reverted to the open technique, which he had since used. He added that a by-pass operation was used in some centres in the United States of America, and that that might be justified for adult patients. Pure infundibular stenosis was rare—he had seen two cases in Sydney—and required a by-pass operation. Secondary infundibular hypertrophy would usually settle down within 12 months of operation, and should be left alone. Occasionally stenosis of the valve ring itself occurred. If that was found at operation it should not be tackled unless the surgeon could proceed with a by-pass operation.

M. T. COCKBURN (Adelaide) asked what should be done with the symptomless child with X-ray evidence of pulmonary stenosis, and when operation should be recommended.

D. STUCKEY (Sydney) commented on the not infrequent occurrence of severe stenosis with no apparent limitation of activity. He said that in conditions such as patent ductus arteriosus, in which surgery produced a normal result and operative mortality was low, then operation was indicated for the symptomless patient. At the moment, the same claim could not quite be made for pulmonary stenosis; but he thought that if a very substantial pressure gradient existed across the valve, then surgery should be performed, even in the absence of symptoms.

I. WALLMAN (Perth) asked whether catheterization was necessary for a child in cardiac failure when the diagnosis was not in doubt.

Dr. Powell commented that two of his patients had not had operative catheterization because, if the diagnosis was incorrect, their prognosis was hopeless in any case.

Symposium on Hyaline Membrane Disease.

KATE CAMPBELL (Melbourne) read a paper entitled "Inhalation of Excess Liquor as a Factor in the Production of Hyaline Membrane Atelectasis" (see page 797).

The Neonatal Pulmonary Hyaline Membrane.

R. A. BARTER (Melbourne) reported a study based on the results of post-mortem examination in 133 consecutive cases of hyaline membrane disease which occurred at the Royal Women's Hospital, Melbourne, during the years 1956-1958. He briefly discussed the current theories of aspiration of amniotic fluid and of capillary exudation as factors causing the lesions seen microscopically in the lung. Dr. Barter said that in 1951, Tregillus had described necrosis of epithelial cells lining terminal bronchioles, and had considered that débris from those degenerate cells passed downwards and was plastered against the walls of alveolar ducts and alveoli in the form of membranes. Little mention had been made of that mode of origin in more recent papers. The results of the present work supported the fundamental idea of Tregillus, but further suggested that degeneration of the cuboidal cells of respiratory bronchioles was mainly responsible for the formation of the membranes, and that material from the terminal bronchioles was only contributory. Stains for elastic fibres showed the characteristic pattern for respiratory bronchioles around spaces lined by hyaline membranes and absence of membranes in the surrounding lung tissue not showing the characteristic elastic tissue pattern. Further, it was emphasized that the alveolar system in the most commonly affected group of babies was far less developed than was usually suggested by writers on the pulmonary hyaline membrane. Studies on the development of the lung were proceeding.

Dr. Barter showed slides to illustrate the following: (i) necrosis of epithelial cells in terminal bronchioles; (ii) the same process in respiratory bronchioles, resulting in the formation of membranes containing only partly degenerate nuclei of epithelial cell type; (iii) the respiratory bronchiole pattern of elastic fibres around spaces containing hyaline membranes; (iv) the formation of membrane-like material in bronchiolar spaces amidst masses of degenerate epithelial cells; (v) varying degrees of degeneration of nuclear structures in hyaline membranes; (vi) probable phagocytosis of membranes by polymorphonuclear leucocytes. He said that the presence of fibrin in the membranes was undisputed, but that it was the major constituent was not supported by conventional staining techniques. Methods used by recent workers to demonstrate fibrin were not quantitative, and claims that fibrin was the major substance present were without foundation. Also, the site of the lesions did not support the hypothesis of an exudative process, since it would be expected that movement of material from the blood would occur into the alveolar components of the lung and not through the epithelial linings of respiratory bronchioles. Dr. Barter further said that microscopic study of the lungs did not support theories which postulated excessive amniotic fluid aspiration, since cell débris from the fluid was seen to any extent in only 31% of the cases.

Dr. Barter finally said that in the present series, nuclear material in membranes was found in 69% of cases and degenerate epithelial cells were found in terminal bronchioles in 70%. Those findings indicated that degenerate epithelial cells largely contributed to the formation of hyaline membranes, and that cellular necrosis was progressive from terminal to respiratory bronchioles. The process of degeneration of the cells lining respiratory bronchioles was apparently a rapid one, and involved considerable swelling of the cuboidal cells and absorption of nuclear material into the protoplasmic mass. It was concluded that hyaline membranes were formed locally in the respiratory bronchioles by the process described, and that an admixture of necrotic cells from terminal bronchioles and of some fibrin was a contributory factor.

THOMAS G. MADDISON (Melbourne) read a paper entitled "The Neonatal Pulmonary Hyaline Membrane". He said that his paper was based on a study of all infants who showed that condition during a three-year period—July, 1955, to June, 1958—at the Royal Women's Hospital, Melbourne. There were 133 infants in the series, and they were the result of 121 pregnancies. He showed that they lived for two to 48 hours, and that three-quarters died in the first 24 hours. Premature infants were the main source of that material, and only 10 weighed more than 5·5 lb. (2·5 kg.). The number of cases decreased as the gestational age increased, and only one infant was post-mature. Of all those infants, 60% were asphyxiated at delivery and took two minutes or more to establish rhythmical respirations. The incidence of complications was high in those pregnancies, particularly multiple pregnancy, preeclampsia, accidental hæmorrhage, placenta prævia and diabetes. The second twin was more likely to develop pulmonary hyaline membrane. Dr. Maddison showed that there was a high incidence of Cæsarean deliveries, and that most of those infants (83%) were asphyxiated at birth.

Although aspiration of amniotic fluid was originally thought to be the factor causing the hyaline membrane, the membranes had even ruptured for over one hour before delivery in 42% of those cases. He showed a diagram of the premature infant mortality, neonatal deaths and cases of hyaline membrane in the different weight groups. There was a high incidence in the 1000 to 2000 gramme ranges. When the incidence was related to the number of live-born infants in each group, there was a very high incidence in the 1000 to 1500 gramme group, which decreased remarkably in the higher weight groups.

Dr. Maddison went on to describe the type of infant who was most likely to develop pulmonary hyaline membrane, and the clinical picture. He then considered the different theories which had been suggested regarding the formation of the membrane. He thought that desquamation of the respiratory epithelium was the only explanation which fitted the facts. He said that Dr. Barter had demonstrated the microscopic findings in those cases. As the infant breathed, the degenerating respiratory epithelial cells were spread against the walls of the terminal bronchioles and alveolar ducts. Any other materials which were present in the air spaces, such as plasma, blood cells or amniotic squamous cells, were incorporated into the so-called hyaline membrane. The progressive downward clinical course followed as the membranes and associated resorption atelectasis developed. The principal associated factors were prematurity, asphyxia at delivery, complications of pregnancy which led to fœtal hypoxia, and maternal diabetes.

Discussion.

CLIFTON WALKER (Sydney) stressed the importance of prevention of the condition. He said that the problem essentially was how to reduce the rate of premature births. Some factors were known; for example, women in a lower economic group had more premature births than those better off financially. Dr. Walker also quoted Simpson, who had found that the incidence of premature births in women smokers was twice as high as that in non-smokers. Dr. Walker asked what treatment was recommended for the condition, and how fœtal distress could be diagnosed early.

CLAIR ISBISTER (Sydney) supported Dr. Campbell's observations that such cases were commonly associated with excessive liquor, but she thought that that was due to retention rather than to inhalation of liquor. Perhaps that was not properly expressed at birth.

JOHN H. COLEBATCH (Melbourne), while agreeing with Dr. Campbell that a search must be made for factors other than excess liquor in the weight group 1250-1500 grammes, stressed the need for extending the search to babies in all weight groups. He said that Dr. Barter's hypothesis seemed well supported by his demonstration of changes in the respiratory epithelium, though that was probably not the whole story. There was a good deal of evidence that circulatory rearrangements after birth produced a transudate into the respiratory tract that was important in that disease. Dr. Maddison's valuable clinical contribution made one look forward especially to his proposed further study of a group of control babies dead of atelectasis without associated hyaline membrane disease. The control of that disease was not likely to depend on prophylaxis alone. Therapeutic measures had already been improved, and a promising trial of fibrinolysins administered into the respiratory tract had begun elsewhere.

Dr. Campbell, in reply, said that preventive measures against inhalation of liquor were being taken. During Cæsarean section the Pash manœuvre was carried out as a routine measure. That was, as soon as the uterus was opened, the infant's mouth and pharynx were sucked out thoroughly, and after delivery he was suspended upside down and the procedure was repeated. Similarly, the procedure was applied to the premature baby as soon as his head was delivered, before he took his first breath. The incidence of hyaline membrane disease had greatly diminished since those precautions had been taken.

Ascorbic Acid and Dried Milk Preparations.

F. W. CLEMENTS (Sydney) read a paper entitled "Should Ascorbic Acid be Added to Infant Dried Milk Preparations?" (see page 800).

D. C. FISON (Brisbane) opened the discussion by commenting on the present position in Brisbane. He mentioned six cases in the previous nine months. One of the patients was a subnormal child of a subnormal mother, and the other five patients were in the 9 to 12 months group. They all presented with a typical story and had typical X-ray findings. They had all been fed boiled cow's milk without orange juice. In all cases, orange juice had been tried by the mother, but rejected by the child. The mothers did not try further with other fruit juices.

ELIZABETH TURNER (Melbourne) said that she and David Farmer in the last five years had seen 256 patients with scurvy, of whom one-third were from New Australian families. She was unable to explain why Victoria should have such a large number. They had tried to increase the health education of the public, and she wondered if Dr. Clements could give some method by which that could be done. She thought that added vitamin C in dried milk preparations might have prevented those cases.

STANLEY WILLIAMS (Melbourne) said he thought that added vitamin C could do nothing but good. Although all children were not fed dried milk preparations, there were many cases of scurvy that could be prevented in children who did have that type of feeding. He wondered about the value of orange juice; it was not always full in vitamin C, and there was the possibility of allergy to it.

J. B. GALBRAITH (Melbourne) spoke of a case of scurvy in a child aged nine months, with radiological evidence of the disease. The child had been receiving 12 mins. of "Pentavite" per day; the mother understood that that was all that was necessary. No orange juice had been given. He wondered if that dose was sufficient—equivalent to 30 mg. of vitamin C—and whether the method of putting drops in the bottle was a satisfactory way of administration.

V. L. COLLINS (Melbourne) asked what percentage of infants were fed on dried milk

Dr. Clements, in reply, said that under 30% at six months were fed in that way, so that the addition of vitamin C to those preparations would add a cover to only a small proportion of the population.

Heredo-Familial Multidystrophic Dwarfism (Cockayne's Syndrome).

PROFESSOR W. B. MACDONALD (Western Australia) discussed a family of five children, three of whom were afflicted with a condition first identified by Cockayne in 1936. Illustrations of the affected siblings in the family described by Cockayne were shown. The siblings, brother and sister, had the following features: a clinical onset in the second year of life after an apparently normal infancy, dwarfism with disproportionately long extremities and large hands and feet, a marked lack of subcutaneous fat of the face with prognathism, sunken eyes and thin nose giving a prematurely senile appearance, and mental deficiency. Other features included kyphosis and ankylosis, sensitivity of the skin to sunlight with pigmentation and scarring, retinal degeneration with pigmentation, optic atrophy, cataract, partial deafness, cold blue extremities, unsteady gait, thickened skull bones and carious teeth. Professor Macdonald said that another family with two affected brothers had been identified by Neill and Dingwall in 1950, and had been reported under the title of "A Syndrome Resembling Progeria". Neill and Dingwall described additional features of coarse tremor, enlargement of the liver and spleen, prominent ears, arteriosclerosis, intracranial calcification and a poor pupillary response to mydriatics. Although intelligence testing classified the patients as idiots, they showed a social adaptation like that of normal young children. Neill and Dingwall noted similarities and differences between those children and those with progeria, and were inclined to relate the two syndromes. Lawson Wilkins, in his atlas of "Endocrine Disorders in Childhood", had included one case of that syndrome and, like Neill and Dingwall, had included it with progeria.

Professor Macdonald went on to describe, with illustrations, the family which he and his co-workers had investigated in Western Australia. The three affected children were a girl, who had died from meningitis at the age of eight years, and two boys still alive, one aged 11 years and the other six years. There were two normal children in the family, one boy and one girl. There was no doubt, as could be seen from the illustrations, that those children were affected by the same condition as were the children in the families described by Cockayne, and by Neill and Dingwall, and the single patient described by Lawson Wilkins.

Additional features described by Professor Macdonald and his colleagues were anidrosis, additional radiological abnormalities of the extremities and vertebræ (notably ivory epiphyses), the relatively high degree of social adaptation compared to the mental ages, transient attacks of unconsciousness, and normal biochemical findings.

Professor Macdonald said that he and his colleagues were not at all impressed with any resemblance to progeria, and he illustrated an example of progeria as a comparison. There was a certain superficial resemblance, but many features were quite distinct. Undoubtedly Cockayne had described an entity, and one in which all affected subjects showed a striking resemblance. It was certainly an heredo-familial abnormality, and almost certainly an inborn error of metabolism. An attempt had been made to delineate that error by biochemical tests. Extensive tests had been carried out, both in the laboratory in Perth and by Professor Dent at University College Hospital, London, but evidence for a recognizable enzyme defect was not forthcoming. That did not negate the likelihood of the condition's being an inborn error of metabolism. To confirm the clinical impression that the syndrome was not related to progeria, plasma samples had been analysed by Dr. Kingsbury at St. Mary's Hospital Medical School, and by Dr. Elspeth Smith of the Courtauld Institute, Middlesex Hospital, London. The investigations included cholesterol, phospholipid and lipoprotein estimations, and the results were unequivocally normal from both laboratories. That was contrary to the abnormal lipo-chemistry of progeria. There seemed to be no justification for including the syndrome with progeria, and for want of a better alternative the eponymous title of "Cockayne's syndrome" seemed as good as any. For those who objected to eponyms, the rather lengthy title of "heredo-familial multidystrophic dwarfism" might be considered, as the disorder was due to some interference with the normal growth and development of many different tissues.

During the presentation, Professor Macdonald illustrated the clinical features of the affected children in the family which he had described, and in addition, the family tree, growth curves and radiological features. Although four generations had been traced, no connexion with the other affected families had been found.

D. B. PITT (Melbourne) asked whether, in view of the attacks of loss of consciousness, electroencephalographic examination had been performed.

Professor Macdonald replied that this had not been performed, as the attacks were not present whilst the patients were under investigation.

Repair of Cleft Palate.

DAVID DEY (Sydney) read a paper entitled "Concepts of Speech Production in Cleft Palate Cases and a Possible Technical Aid" (see page 801).

PROFESSOR ORVAR SWENSON (Boston) agreed that lengthening of the palate was not important in the production of speech. He said that that was illustrated by the failure of the Darance operation, which was designed to produce long palates. A mobile palate was essential. In the U.S.A., a less radical operation was used, with a less lengthy incision and no flaps.

DOUGLAS STEPHENS (Melbourne) asked whether the flap stayed in place for good, and whether there was any virtue in the Denis Browne circumpharyngeal stitch.

Dr. Dey replied that he thought the excellent healing achieved by Denis Browne was due to the skill of the rest of the operation rather than to the circumpharyngeal stitch.

SIR KENNETH FRASER (Brisbane) agreed that mobility of the palate was a vitally important factor. He said that complete closure of the naso-pharyngeal isthmus was not necessarily a *sine qua non* for perfect speech. The ability of mother and patient to persevere in efforts to improve consonantal speech were of tremendous importance. Sir Kenneth Fraser then mentioned a patient on whom he had operated 23 years earlier, who had returned a week before. The patient had a very definite hole at the junction of the hard and soft plates and a wide naso-pharyngeal isthmus, yet spoke perfectly. He was the type of boy who was determined to speak perfectly, and had had a fight (which he won) on his first day at school because of the slurring of his speech. Such a case was admittedly unusual; but it could happen, provided that the palate was mobile and persisting speech reeducation had been maintained. With regard to the tying of blood vessels, Sir Kenneth Fraser

said that he thought that was all right provided there were no long flaps. If there were, the vessels must be retained and mobilized by chiselling out of the posterior wall of the palatine foramen. In the Wardill operation, which he had used for 25 years, there was no scarring along the line of the lateral incision. The two places in the body in which healing could occur without scarring were the mucosa of the palate and the penile skin. In palatal surgery scarring, if any, was in the mid-line, and that was what affected mobility. Scarring of that nature could be avoided if mobilization of the flaps was effected so that they fell together without tension.

Congenital Arachnoid Cysts.

M. SOFER SCHREIBER (Sydney) read a paper entitled "Primary Congenital Arachnoid Cysts" (see page 802).

C. A. RIGG (Sydney) asked whether questioning of mothers had elicited any noxious influence about the fourth month of pregnancy.

Dr. Schreiber replied that it had not.

Recent Advances in Biochemistry.

PROFESSOR A. H. ENNOR (Canberra) spoke on "Recent Advances in Biochemistry". He showed how clinical biochemistry was becoming able to explain the signs and symptoms of clinical medicine in terms of chemistry and enzyme systems. He went on to explain the enzyme defects present in galactosæmia (galactose phosphateuridyl transferase) and "physiological jaundice of the newborn" (glucuronosyl transferase).

A discussion on the pathways involved in the oxidation of the glycine and threonine molecules ensued. Finally, as a piece of pure research by his own laboratory, he discussed the significance of the isolation of a dextro-rotating amino-acid (d-serine) from lombricine, a substance playing a central role in muscle metabolism in the earthworm.

Imperforate Anus.

F. DOUGLAS STEPHENS (Melbourne) read a paper entitled "Imperforate Anus" (see page 803).

PROFESSOR ORVAR SWENSON (Boston), in opening the discussion, commended Mr. Stephens for his careful study. He said that their experience in Boston with low-level lesions coincided with his. They gave the best results, and that was probably related to the fact that the various components of the sphincter mechanism were more complete with the low-level lesions. Professor Swenson said that they had followed 64 patients with lesions at various levels for a period of at least three years, and the results were satisfactory in the low-level lesions and unsatisfactory in the high-level lesions. In order to elucidate the position, Dr. John Scott, working in his department, had performed a series of pelvic dissections on subjects with high-level lesions. He had been particularly concerned with the status of the parasympathetic and sympathetic nerve supply. He had found that, even in the high-level lesions, the autonomic system was present. The more extensive operative dissection required in those cases probably damaged the nerve supply to a considerable extent. Surgeons in repairing that lesion should strive to disturb the neurogenic mechanism as little as possible. That could be achieved by dissecting the bowel as carefully as possible, and by leaving attached to it as much areolar tissue as possible. There was a limit to what surgery could achieve. Even with low-level lesions it was rare to see a child who was perfectly continent of fæces—one who could withstand a brisk bout of diarrhœa. It had to be remembered that the normal sphincter was a very complicated neuro-muscular mechanism, and that, in addition to the sphincter muscle itself, there was a complicated sensory system connected with the sphincter by a complicated neurogenic mechanism. Sufficient thought had not been given to that neurogenic mechanism, and one could not picture moderately extensive imperforate anus without a disturbance of those neurogenic elements. Many physicians did not realize that the chief problem in cases of imperforate anus of the high-level variety was colonic inertia, and that the soiling that was so common was due to fæcal impaction. Because that condition was uncommon, the physician was inclined to be unaware of the various tricks in the post-operative management of those patients. The objective was to secure sufficient colonic emptying to prevent impaction and subsequent diarrhœa and soiling. There were two ways of doing that. The first was by bowel irrigations, which the mother was taught to perform religiously once a day or at least on alternate days. The second method was the use of a rectal suppository ("Durolax"), which would induce intestinal peristalsis. That was simpler for the mother than irrigation; however, it did not invariably work. Provided that a programme could be worked out which ensured that the colon was emptied at intervals of 24 to 48 hours, there would be no soiling of any consequence and the child could live a fairly normal life. In summary, Professor Swenson said that the two important principles in the management of the condition under discussion were: (i) surgery should be carefully performed, and every small advantage presenting itself anatomically should be taken; (ii) adequate follow-up care was most important, to prevent massive impaction and all the problems attendant on that complication.

T. Y. NELSON (Sydney) said that there was a superficial resemblance between the condition and megacolon, and asked whether Professor Swenson had seen a similar condition of colonic inertia to any extent after recto-sigmoidectomy in megacolon.

Professor Swenson, in reply, said that the result of recto-sigmoidectomy for aganglionic megacolon was about 98% effective. In 2% or 3% of cases colonic inertia, or more commonly chronic diarrhœa, was a cause of poor results. It was not uncommon to have a patient referred who had had an imperforate anus repaired, and who had developed massive impaction, and with it a real megacolon. The question arose whether that was to be considered in the same category as the aganglionic megacolon. Professor Swenson said that a number of those patients had undergone biopsy, and as yet they had not found one with a defect in the Auerbach's plexus ganglion cells. For that reason, resections on patients with imperforate anus and megacolon had not been successful. The troublesome types of complication that they had found after operation in cases of aganglionic megacolon were recurrent diarrhœa and acute bouts of enterocolitis. That was a serious problem, and it was not well appreciated. The patient might have a slight elevation of temperature at the beginning of such an attack: he vomited and passed a few loose stools. Then his abdomen became massively distended, and he had paralytic ileus. That condition was extremely dangerous to the patient, and might be the cause of sudden death. Should the patient survive the acute attack, the condition might become chronic, and the child's abdomen would remain distended and he would pass foul liquid stools. Often he became incontinent of fæces. The cause of that enterocolitis was unknown. It might be related to the fact that those patients still had partial obstruction because they still had a short aganglionic segment of rectum in place and also had a tight aganglionic sphincter. Certainly dilatation of the sphincter was effective in the treatment of those patients. Professor Swenson said in conclusion that they had recently used sphincterotomy, and had been most pleased with the results.

E. STUCKEY (Sydney) asked Dr. Stephens in what percentage of cases in which high and middle lesions were present he used the approach from the sacral region, and how often it was necessary to perform an abdominal incision in order to free the bowel. He also asked whether it was possible to perform the operation from above without the sacral approach. Dr. Stuckey finally asked Dr. Stephens in what percentage of middle and high lesions he achieved continence of fæces, and for what period of time he had followed up the patients.

Dr. Stephens, in reply, said that when the bowel descended to the level of the pubo-coccygeal line, operation could be performed from the sacral regions in males. However, in females there might be a strong band-like attachment from the bowel to the cervix, often preventing adequate mobilization if the sacral approach only was used. If the bowel did not reach the pubo-coccygeal line, then operation must be performed by the sacral and abdominal routes. The intermediate level abnormalites were hard to mobilize from the perineal approach, and stricture formation was prone to occur. Operation was probably best by the sacro-coccygeal route. Dr. Stephens then said that he did not know whether operation from above only took sufficient care of the puborectalis sling. That could perhaps be done more readily in older and larger children than in neonates. In the intermediate and low-level lesions, a respectable degree of continence was produced. The patients with high-level lesions required no more than an occasional change of underpants. Rectal examination gave a good idea of the degree of continence; if a thick collar of puborectalis was present, continence was good.

Abnormalities of Ureteral Peristalsis.

PROFESSOR ORVAR SWENSON (Boston) read a paper entitled "Studies of Normal and Abnormal Ureteral Peristalsis" (see page 805).

P. GRANT (Brisbane) asked whether Professor Swenson had carried out any peristaltic studies after a course of triple micturition.

Professor Swenson replied that he had, but that it was difficult to say just which factor in the management had improved the condition. Triple micturition was good, provided that there was no residual urine. It had been thought at first that when there was reflux, there would be no peristalsis; but that was not so. A patient with reflux who had vigorous ureteric peristalsis could be given a good prognosis.

F. D. STEPHENS (Melbourne) asked Professor Swenson if he could demonstrate the normal tracing of the pelvis of the kidney, as opposed to the ureter.

Professor Swenson replied than an undulating type of tracing was obtained from the renal pelvis. The pelvis produced pressure changes of only 3 to 5 cm. of water. Ureteral contractions were considered due to a simple stretch phenomenon rather than to a neurogenic mechanism. The ureteric tracing showed regular peaks of high pressure; they became more peaked and the pressures higher as one proceeded down the ureter. The undulating pelvic contractions appeared to initiate peristaltic waves. There was little that could be done to dampen ureteral peristalsis, apart from altering the urinary output. No drugs, anæsthetic agents included, demonstrably affected ureteral peristalsis.

SIR KENNETH FRASER (Brisbane) asked whether the relationship was known between the pressure in the normal bladder and the pressure in the lower end of the ureter during micturition. He wondered whether cases of reflux were due primarily to an accentuation of the difference between those pressures, or to a deficiency in the neuromuscular closing mechanism at the uretero-vesical junction.

Professor Swenson replied that he thought both factors were important, though it was very difficult to prove. In a child whom he had followed for some years, a sudden breakdown had occurred at the uretero-vesical junction. Whether that was defective to start with was not known. In some patients reflux was present from the beginning, and there it appeared that there was a defect in the mechanism. The best example of that condition was the patient with exstrophy of the bladder. There no back pressure was present; but almost uniformly reflux occurred on closure of the bladder. Therefore one had to assume a congenital defect of the junction as well. Professor Swenson said that combined pressure values were very difficult to obtain, and he therefore could not answer Sir Kenneth Fraser's first question.

R. SOUTHBY (Melbourne) commented that Professor Swenson's work stressed the importance of attacking urinary infections from the first acute episode.

W. GRIGOR (Sydney) said that as a student he had been taught that morphine by itself was not the drug of choice for renal colic. It should be combined with atropine, or pethidine should be given instead. He asked whether Professor Swenson's work invalidated that theoretical concept. Dr. Grigor also asked whether it was correct to assume that the absence of ganglion cells in the normal ureter implied that drugs would fail to alter ureteric contractions. He wondered whether there would be any hope of using a parasympathomimetic drug to stimulate ureteric contraction, encourage drainage and thus help in the treatment of chronic renal infection.

Professor Swenson replied that morphine and other drugs could be given without affecting the tracing. There was no demonstrable evidence that those drugs worked, and certainly they did not work too well clinically. No ganglion cells could be demonstrated in the ureter, except perhaps right at the lower end—and those probably belonged to the bladder. The behaviour of the smooth muscle of the ureter appeared different from that of other smooth muscle. The mechanism appeared more like that of heart muscle, in which the impulse spread through the heart mass rather than by a neural mechanism.

A. M. CLARKE (Melbourne) asked what chemotherapy should be used in the management of chronic pyuria.

Professor Swenson, in reply, said that it was wrong to give long courses of broad-spectrum antibiotics. An acute attack should be vigorously treated for four or five days; but such vigorous treatment should not be carried on in the chronic condition, as that was commonly a mixed infection, and overgrowth of resistant organisms, such as Proteus, was likely to occur. Professor Swenson said that in chronic cases he recommended the use of urinary antiseptics, such as "Gantrisin", "Furadantin" and "Mandelamine" in very small doses—for example, 250 mg. of "Gantrisin" three times or twice per day. That maintenance dose should be continued, and the physician must be patient, as improvement might be imperceptible for a long time. Professor Swenson went on to say that he had had one patient taking "Gantrisin" for eight years. The improved ureteral peristalsis and the appearance of the children had convinced him of the effectiveness of that treatment. Unfortunately he had no bacteriological data to support his other observations. Various urinary antiseptics might be given in rotation, each for a fortnight at a time.

Neonatal Meningitis.

ELIZABETH K. TURNER (Melbourne) read a paper entitled "Neonatal Meningitis" (see page 806).

STANLEY WILLIAMS (Melbourne) emphasized the poor prognosis of the illness, many of the babies who recovered being left with sequelæ such as hydrocephalus.

PROFESSOR ORVAR SWENSON (Boston) asked whether any of Dr. Turner's cases were post-operative. He said that not infrequently he had seen meningitis after operation in a baby not progressing satisfactorily, often with no marked signs or symptoms.

J. W. PERRY (Melbourne) commented that the figures at the Royal Children's Hospital, Melbourne, were similar to those reported by Beveridge in Sydney, with regard to both the incidence of enteric organisms and the mortality figures. At Great Ormond Street, considerable emphasis was placed on the association with urinary tract abnormalities, but at the Royal Children's Hospital, Melbourne, the incidence of renal abnormality was low. He wondered what Dr. Turner had found in that regard. The commonest autopsy finding in his experience was pyocephalus.

KATE CAMPBELL (Melbourne) mentioned a baby, aged 36 hours, with meningitis following premature rupture of the membranes, who had done very well.

D. G. HAMILTON (Sydney) asked whether the introduction of corticosteroid therapy had made any difference in those cases.

C. A. RIGG (Sydney) asked how many of the deaths were due to streptococcal infection, and, if the patients had not died, how had they responded to treatment. He added that in Boston they had found streptococcal meningitis in the newborn very difficult to treat, and those cases were almost invariably fatal.

SIR KENNETH FRASER (Brisbane) suggested that affected babies should be moved round a lot and turned upside down every few hours. He said that Sir Hugh Cairns had emphasized constant movement to prevent blocks occurring.

Dr. Turner, in reply, said that none of her cases occurred after operation. All babies with gross abnormalities of the central nervous system and of the urinary tract were excluded. The poor prognosis was difficult to explain. The incidence of streptococcal infection varied in different surveys, being five out of the 45 in the present series. Corticosteroids were not used in any of those cases because of the known increase of susceptibility to infection. No special movements were prescribed for the babies, simply unrestricted movement as with an ordinary neonate.

Enteric Virus Infection.

JOHN FORBES (Melbourne) read a paper entitled "Diagnosis of Enterovirus Infections" (see page 808).

Disordered Physiology Following Bowel Resection in Infancy and Childhood.

CHARLOTTE ANDERSON (Melbourne) read a paper on "Disordered Physiology Following Bowel Resection in Infancy and Childhood". She based her remarks on observations made on four patients who had been referred by surgeons, and who had one feature in common—the surgical removal of some part of the gastro-intestinal tract. Those patients illustrated some of the difficulties encountered in the restoration of normal physiological function after such operations.

The first patient, A, a boy, was mentioned briefly, largely for interest. At the age of two years and three months, in Holland, A. drank some ammonia, and subsequently had four-fifths of his stomach removed with what was described as an ante-colic long-loop anastomosis performed between the remnant of the stomach and the jejunum, a blind but self-emptying duodenal loop being left. Although the child was mentally retarded, he remained fairly well until the age of six years, when he was noticed to become increasingly pale and to have attacks of diarrhœa. At six years and four months he was examined at the Royal Children's Hospital, Melbourne; his hæmoglobin value was then 26%, a blood film showed macrocytosis and his bone marrow was megaloblastic. After transfusion, other investigations were performed. The

stools were pale and shiny; fat absorption was only 76% instead of 90% to 95%. He had histamine-fast achlorhydria, the pancreatic enzymes were only slightly reduced, and the bacterial flora of the upper part of the small intestine was normal. He therefore showed an adult type of post-gastrectomy syndrome, with megaloblastic anæmia and steatorrhœa. At that stage he was not given continuous vitamin B_{12} therapy. His blood remained normal for one and a half years, but then he returned from the country very anæmic again. On that occasion a vitamin B_{12} absorption test with radioactive cobalt (the Schilling test) showed very poor absorption of the vitamin. There was a good reticulocyte response to vitamin B_{12}, and his hæmoglobin value rose. At the time of the meeting he was being kept on monthly injections of vitamin B_{12}. Dr. Anderson said that fortunately removal of such large amounts of the stomach in childhood was rare; but it appeared that a child could develop a similar post-gastrectomy syndrome to that seen in adult life. The feature of interest was the length of time after the first operation before anæmia was apparent—almost four years. That, of course, had also been noted in adults, in whom anæmia might take up to five years to appear.

The remaining cases were examples of resection of extensive portions of small intestine with anastomoses at different points. The patients had been referred after the surgeons had carried out the necessary operation, usually with the request: "What do we do now about the feeding of this child?"

Dr. Anderson said that very little information appeared in the literature about what happened later to children who had had extensive small-bowel resections, although a copious but rather confusing literature existed regarding adult patients. Broadly, it had been found in adults that 50% of small bowel might be removed with return to normal function and health. All but about 18 inches might be removed with subsequent survival, but only on a modified diet—one that had a low fat content, and a high content of protein and calories. Removal of amounts greater than that—leaving less than 18 inches—had not resulted in survival of the patient beyond two or three years. Pilling and Cresser in 1957 had reported two babies who lost all but about 28 cm. of their small intestine, and in a follow-up period of two to four years had progressed satisfactorily, but could tolerate only a diet of low fat content. At the outset, caloric content and fluid intake had to be high. Fat was able to be introduced slowly into the diet after about two years of age.

Dr. Anderson then said that the prognosis and type of treatment after small-bowel resection did not depend only on the length of bowel left *in situ*, but on a number of factors. First, the initial pathology of the lesion was important, and in its turn sometimes determined the state of the bowel left *in situ*. For instance, the small bowel left behind might not be structurally normal; or during the period of obstruction before operation, permanent damage might have occurred to the mucosa, leading to subsequent malabsorption. The type of operation which had to be performed might lead to subsequent alterations of function of the remaining small bowel—for instance, the presence or absence of the ileo-caecal valve, the creation of a blind loop, or subsequent dilatation of the small bowel in the region of the anastomosis leading to stasis of its contents. An intact large intestine was probably important, and that could adapt itself to the altered amount and composition of the fluid which entered it from the small intestine, and to alterations in its rate of entry.

When the post-operative management and progress of such patients were considered, some attention and thought should be given to the normal functions of various parts of the intestine. The stomach received food, initiated digestion, allowed discharge of contents into the duodenum at a certain rate, and added "intrinsic factor" to the intestinal secretions. It absorbed alcohol, and possibly a little water and sugar. In the small intestine digestion continued, and it was presumed that the rate at which food entered it, its total length, and the rate of passage of fluid along it all played a part in the completion of digestion by pancreatic and other enzymes. Protein, sugar and fat were absorbed from the small intestine, the former mainly in the upper small intestine and the latter mainly from the lower portion. Vitamins and iron were also absorbed, iron in the upper part and vitamin B_{12} in the lower part. Fluid and electrolytes were probably partly absorbed from the lower part of the ileum. The ileo-caecal valve allowed passage into the colon at a certain rate. In the colon water and electrolytes were further absorbed. The flora of the small and large intestine also differed greatly, the small intestine being relatively sterile and the large intestine having a profusely fæcal growth throughout its extent. Stasis occurring anywhere along the small intestine could change the flora of that area from that of relative sterility to a profusely fæcal growth just like that of the large bowel, with consequent alteration of some of the functions of the small intestine.

Dr. Anderson then described the post-operative clinical progress of several of the patients referred. The first was B., a girl, who had presented at the age of one day with intestinal obstruction, and at operation was found to have atresia of the ileum. Ileostomy and resection of the terminal portion of the ileum were performed and a few days later an end-to-side anastomosis of the ileum to the transverse colon was carried out. The baby, after recovery from operation, was given a dilute sweetened condensed milk feeding; but severe diarrhœa ensued, and gradually over succeeding weeks she developed abdominal distension and a ladder pattern on the abdominal wall. During the first four months many types of feedings were tried, but no progress in weight or clinical condition was made. Diarrhœa and distension continued, and a barium meal X-ray examination revealed great dilatation of part of the small intestine. Laparotomy was performed, and the terminal 18 inches or so of the small bowel were found to be greatly dilated, but the anastomosis was patent and large. The caecum seemed of normal size. The dilated small bowel contained much fluid growing a profuse fæcal flora. Resection of the anastomosis, the dilated small bowel and the right side of the colon was performed, with re-anastomosis, end to end, of the ileum to the remaining colon. However, subsequent progress was equally poor, diarrhœa continued and gross steatorrhœa was shown to be present. Pancreatic enzymes were normal, but small intestinal content still grew a fæculent flora. A barium meal X-ray examination again showed dilatation of the terminal portion of the small bowel. By this time it was considered that about half the small intestine had been removed, and removal of any more was not contemplated.

That was the stage of the child's first referral, and she was placed on a diet of very low fat content, pancreatin was added and a programme of changing antibiotics in small doses was instituted, three or four days of each antibiotic. At that stage the baby at last started to gain in weight, her stools were much fewer and firmer, and she was able to be discharged from hospital at the age of nine months, weighing 12 lb. Her subsequent progress had been fair, but she had not really grown at a rate up to the 10 percentile for Australian children. Whilst she was kept on a reduced fat diet with pancreatin and antibiotics, the growth rate, intestinal absorption and character of the stools were better, although not normal; but when pancreatin and antibiotics were ceased, the growth rate fell off, diarrhœa recurred, steatorrhœa was worse and anæmia of a macrocytic type occurred. The Schilling test showed poor absorption of vitamin B_{12}. It seemed that a number of factors might be contributing to the child's difficulties. First, the question arose whether the small bowel proximal to the atretic portion was neurologically normal, owing to its persistent tendency to dilate subsequent to each operation, or whether the type of anastomosis performed had anything to do with that. Dilatation of small bowel led to stasis, growth of fæcal flora and malabsorption. The shortened bowel might also lead to malabsorption of vitamin B_{12} with subsequent megaloblastic anæmia. The absence of the ileo-caecal valve and half of the colon probably aggravated the tendency to diarrhœa, owing perhaps to inadequate absorption of water from the terminal portion of the small bowel and the remaining large intestine.

Dr. Anderson then said that the next patient, C., a boy, had made better initial progress, profiting from some of the mistakes in the earlier treatment of B. C. on his first day of life had received an exchange transfusion; on his third day of life intestinal obstruction occurred, and on the fourth day a laparotomy was performed, with removal of about half of the small intestine and an end-to-side ileocolostomy for a mesenteric thrombosis which had caused bowel gangrene. The caecum was left *in situ*. During the post-operative period he had numerous fluid stools, but on a greatly increased fluid and caloric intake and a feeding of very low fat content he gained weight very satisfactorily and was able to be discharged from hospital. His subsequent progress was good, his growth being at first just above the 10 percentile until 12 months. His growth then slowed up until 15 months, and during that time he was noticed to become paler. His bowel motions were never normal, but they were not troublesome until during the latter three months, when an effort was made to increase the amount of fat in the diet. That was

not persisted with; but nevertheless, at the age of 15 months, he suffered acute diarrhœa with gross fluid and weight loss and collapse. He was resuscitated, but during the subsequent weeks was unable to be established on a feeding. Profuse diarrhœa continued, and he had no less than six intravenous infusions. On his admission to hospital on that occasion he was found to be suffering from megaloblastic anæmia with a hæmoglobin value of 48%, of which there had been no obvious sign at the age of 12 months. His abdomen was distended, a ladder pattern was visible and fluid splash was audible. A barium meal X-ray examination showed dilatation of the terminal portion of the small bowel and the whole of the large bowel, with gross dilution of the barium indicating large fluid content. On entering the large bowel, the barium passed first into the caecum, where it lingered for several hours before passing on along the colon. At that stage, when the child's life was being despaired of, the surgeon reopened the abdomen, undid the ileo-transverse anastomosis and placed the terminal portion of the ileum back in the caecum. At operation the terminal portion of the small intestine was grossly dilated and full of fluid containing a profuse fæcal flora. Since that operation the child had been considerably better and was now gaining weight, although the bowel actions were still loose and large, and fat absorption was almost completely absent. Both those disorders had been partially helped by the giving of pancreatin with meals, and antibiotics in small doses in a rotating series. He still required a large fluid and caloric intake. The anæmia had partially responded to vitamin B_{12}, the absorption of which was shown to be low. Dr. Anderson commented that that child also had the combination of short small intestine, dilatation of the terminal portion and stasis with bacterial proliferation, giving similar clinical features to a blind-loop syndrome. It was thought that incorporating the caecum back again into the continuity of the intestinal flow had greatly helped him, although the absence of the ileo-caecal valve was still contributing unfavourably. He certainly lost his abdominal distension and ladder pattern and fluid splash after the second operation, but absorption was still greatly impaired.

The fourth patient was D., an unfortunate young girl, who two years earlier, at the age of 14 years, suffered a volvulus of the whole of her small intestine, resulting in gangrene, and the removal of all but the proximal three inches of the jejunum and the distal three inches of ileum—six inches in all. She had made a fairly good recovery in the early stages, but her weight before illness was seven stone, and after operation it was four stone ten pounds, and during the succeeding two years had remained at or about that figure, there being no increase at all despite numerous measures. At first she had passed numerous fluid stools, but they had gradually abated to one or two very large ones each 24 hours. Seven months after operation, when she was first referred, X-ray examination of her gastro-intestinal tract revealed considerable dilatation of the stomach and the large bowel, features which had been shown to occur in other case records in the literature. That apparently helped in the absorption of fluid and in decreasing the number of evacuations per day. Although the small intestine was shown to be extremely short, the presence of the ileo-caecal valve helped to control the flow from stomach to large bowel. Other investigations carried out at that time showed normal blood chemistry and hæmatology. Fat and nitrogen balance studies showed severe defects, fat absorption being only 23%, and nitrogen absorption 43%. It was noted that when fat intake was higher absorption was less, and absorption of nitrogen was also less, a fact observed by others in the literature. Dr. Anderson said since that time the girl had been kept on a diet of low fat content and high protein and simple sugar contents, with frequent feedings. Adjuncts such as fat-emulsifying agents of the Tween 80 type, slowing agents and pancreatin had all failed to help her absorption. Vitamin B_{12}, vitamin B complex and others had all been given regularly, with maintenance of a normal blood picture, and during the past 18 months the serum chemistry had remained remarkably normal. However, she had gained no weight at all, her clinical condition had worsened, resulting in periods of great weakness, sometimes hypoglycæmic episodes, sometimes episodes of unconsciousness or great sleepiness. A further period of hospital care with intensive feeding and rest had improved her a little, but it was thought that her prognosis was poor. Only one other record of a patient who had only six inches of small bowel remaining had been found. That case had been reported by Jackson and Linder of South Africa, and the clinical course was very similar to that of D., until the patient gradually faded out from inanition three and a half years after operation. It seemed that the girl D. simply had not enough small intestine to enable protein absorption to be adequate for growth. However, it was of interest to note the maintenance of relative homœostasis in her electrolyte pattern. Good absorption of fluid and electrolytes from the large bowel and the presence of the ileo-caecal valve and large bowel continuity were probably important in that connexion.

In summary, Dr. Anderson said that the cases presented illustrated some of the difficulties encountered in the medical management of children after gastro-intestinal resections. No doubt there were many patients who did not have those troubles; but study of those patients had led to some recommendations in reference to both the immediate operative period and the long-term management. First, in regard to the operation, the surgeon had to deal with conditions as he found them within the abdomen; but a plea could be made for leaving the ileo-caecal valve if possible, for preserving intestinal continuity and absence of loops in which stasis could occur, and for retaining as much colon as possible. Dr. Anderson asked for comments from the surgeons on the type of anastomosis—end-to-end or end-to-side. She postulated that the end-to-side anastomosis might deleteriously affect motility. In the immediate post-operative period, fluid and caloric intake must be high and fat intake low, and one must not be deterred by the fact that bowel actions were abnormal or frequent. In long-term management, the diet should have a low fat content particularly. Extra pancreatin might help, as excessive rate of passage did not seem to give the natural pancreatic juices adequate chance to be effective. No other adjuvants, such as slowing agents, emulsifying agents, protein hydrolysates, etc., had been found of any value. If stasis or "blind loop" syndrome occurred, antibiotics given in small doses and changed every four to five days might be helpful in depressing bacterial growth in the small intestine. Attention should be paid to the development of anæmias, which might not occur for a considerable time—up to years after the initial operation.

PROFESSOR ORVAR SWENSON (Boston) said that he had been pleased to hear Dr. Anderson's recommendations. For years he had preached that an abnormal situation should be restored as close to normal as possible. Some years earlier he had published a paper stressing the importance of end-to-end anastomosis. He said that that would not eliminate problems, but would prevent some and obviate the necessity for subsequent operations.

S. WILLIAMS (Melbourne) asked whether the role of antibiotics was in suppressing abnormal bowel flora or in reducing respiratory infections.

Dr. Anderson said that the doses of antibiotics used were aimed at suppressing the abnormal flora in the small intestine. Each antibiotic was used for only a few days at a time and then changed. The progress of the second patient particularly was better when antibiotics were used.

F. D. STEPHENS (Melbourne) asked for more information on the "blind loop syndrome". He said that he had operated on two of those patients, and the easiest operation at the time was to anastomose the closely approximating terminal portion of the ileum and the transverse colon. Now he had retrieved the situation as far as possible technically, but wondered whether he should have done anything further at the second operation, particularly on the third patient.

Dr. Anderson said that in the third case, the second operation appeared to have been life-saving. At least after that the boy had not needed further intravenous therapy, and although absorption was still extremely poor, perhaps that was due to a too short small bowel or to damage to the absorptive surface during an earlier stage. In the so-called "blind loop syndrome", stasis of contents in some portion of the small intestine occurred with bacterial proliferation leading to competition for vitamin B_{12} and malabsorption. In some of those cases, dilatation of the terminal portion of the small intestine had produced a similar state of affairs. There was certainly stasis of fluid, with bacterial proliferation and poor absorption of vitamin B_{12}. She did not know whether leaving the caecum and ascending colon out of continuity with the intestinal stream played any part in that, as it should theoretically be a self-emptying loop. She thought, however, that it might not actually be so when an end-to-side anastomosis was made to the transverse colon. Events in the third case seemed to support that.

R. SOUTHBY (Melbourne) asked whether the first patient with histamine-fast achlorhydria required any dilute hydrochloric acid to keep the bowels normal.

Dr. Anderson said that diarrhœa was not now a troublesome feature after vitamin B_{12} therapy, so as yet no hydrochloric acid had been given.

Fatal Collapse During Exchange Transfusion.

S. E. J. ROBERTSON (Sydney) read a paper entitled "Fatal Collapse During Exchange Transfusion" (see page 809).

KATE CAMPBELL (Melbourne) thanked Dr. Robertson for a controversial paper, and said that in her experience she had found Wiener's method dangerous, it often being impossible to correlate bleeding with giving. Out of 322 exchange transfusions performed since 1952 by Dr. George Champion, of Melbourne, there had been only one fatality, which was due to the blood being too hot. Dr. Campbell said that the temperature should never be above 90° to 95° F., and that the rate of exchange of 100 ml. in six minutes seemed safe, 10 ml. at a time being exchanged in full-time babies and 5 ml. in premature babies. Dr. Campbell went on to say that she had never worried about venous pressure and had not bled the infants first, but gradually built the balance up or down on occasions during the procedure. She gave calcium only if the baby was restless and crying a lot. The infants were quieted with a comforter. She believed it to be most important that the exchange should be carried out by a team, with one transfusing and with a stethoscope on the baby's chest. If the heart sounds changed in cadence, then it was time to stop.

H. RISCHBIETH (Adelaide) spoke of the last 125 exchange transfusions he had performed, in which there had been no fatalities. He agreed that the temperature of the blood used should not be above 90° F., and that it should not be more than one day old, as the free potassium rose rapidly after 24 hours' storage.

B. W. NEAL (Melbourne) asked whether if, as Dr. Robertson had said, the venous pressure reading could be misleading, reading the pressure after every 100 ml. gave any useful information.

D. G. HAMILTON (Sydney) said that he supported Dr. Robertson, and that he thought that the rate of exchange of 100 ml. in six minutes was a fast gallop and very dangerous.

M. T. COCKBURN (Adelaide) said that he supported Dr. Robertson, and that it was not speed that was essential in such an important procedure, but the safety of the patient.

M. J. ROBINSON (Melbourne) disagreed. He considered that 100 ml. in six minutes was not excessive.

Dr. Robertson, in reply, said that he agreed with Dr. Campbell that Wiener's method was difficult to control, and it had been used only for the third and fourth exchanges. When the cord had fallen off, it was quite easy to find the umbilical vein by making a small incision above the umbilicus, isolating the umbilical vessels and incising their fibrous sheath. However, on one occasion there must have been a small umbilical hernia within that sheath, because while he was searching for the vein a large length of small bowel suddenly prolapsed into the wound, causing great consternation and considerable difficulty. Since that occasion, when the umbilical site was no longer available, a catheter had been inserted into the upper end of the great saphenous vein where it joined the femoral vein, and the catheter was then advanced into the inferior vena cava. That was a very satisfactory method, but it was technically difficult in a small infant. Preliminary reduction of the blood volume was vital in the hydræmic, severely affected infant. Dr. Robertson said that continuous listening to the heart sounds was excellent, but meant an extra doctor. Many exchanges were carried out only with nursing help, and he believed that watching the colour of the withdrawn blood and the condition of the infant was more practical. To those speakers who said that a rate of exchange of 100 ml. every six minutes was safe, he could only say that he emphatically did not agree. The maintenance of the temperature of the blood at 100° F. was considered quite safe by the Blood Transfusion Service of the Australian Red Cross Society (New South Wales Branch). It had to be realized that the blood cooled while it was in the filter and tubing between bottle and syringe. In conclusion, Dr. Robertson said that he would like the audience to realize that exchange transfusion was carried out by individual doctors at odd hours in hospitals where help was unskilled. Ideas as to technique would vary; but the ideas in his paper were the result of considerable experience. To summarize, he had learnt to use freshly collected blood, to work with a negative balance suitable for the condition of the patient, to watch the withdrawn blood and the condition of the infant and, above all, not to hurry.

Staphylococcal Empyema.

E. S. STUCKEY (Sydney) read a paper entitled "Staphylococcal Empyema" (see page 811).

V. L. COLLINS (Melbourne) emphasized the increasing problem that staphylococcal empyema had been presenting in recent years. He said that in 1957, of 40 cases of empyema at the Royal Children's Hospital, Melbourne, 35 were staphylococcal in origin, and a majority occurred in infants aged under one year. In 1958, 30 patients suffering from staphylococcal empyema were treated. A comparison had been made of the number of cases of staphylococcal pneumonia occurring in the periods 1937-1938 and 1947-1948, when there were usually three to four patients each year suffering from staphylococcal empyema. There had been a steady increase in the number of patients since 1953, and that increase in recent years appeared to be considerably greater than the increase in the number of infants in the population, which had approximately doubled since 1937. When diagnosis was made early, the majority of patients responded promptly to antibiotic therapy and intercostal drainage with an underwater tube.

P. G. JONES (Melbourne) agreed particularly with Dr. Stuckey's criticism of a small cannula. He said that any catheter large enough to produce adequate drainage would be compressed by the ribs of a small baby. That left two alternatives: (i) rib resection and (ii) the use of a metal catheter forced between the ribs and holding them apart—e.g., the Reynolds tube. That method was of value for the very sick, toxæmic infant, though rib resection and drainage would usually be required later. Fluorographic examination of the cavity might be helpful—a No. 1 polythene catheter was inserted and allowed to coil up in the bottom of the cavity, "Lipiodol" being injected on withdrawal.

PROFESSOR ORVAR SWENSON (Boston) stressed the dangers of early open drainage. He said that early rib resection (before mediastinal fixation) produced an impossible physiological situation. The early institution of adequate intercostal drainage made the operation of decortication rare. Professor Swenson reinforced Dr. Stuckey's plea for early surgical consultation. In all cases of staphylococcal pneumonia, equipment for thoracic aspiration should be by the bedside. He wondered if Dr. Stuckey favoured digitalization of some of the very sick patients.

R. GODFREY (Perth) supported very strongly the remarks about the rare necessity for rib resection. He said that early drainage might be necessary to save life. He also stressed the importance of having equipment by the bedside so that aspiration could be performed at a moment's notice by anyone present, not necessarily a surgeon.

S. W. WILLIAMS (Melbourne) commented that death might occur early from toxæmia, and that the surgeon should always work closely with the physician. He held that gamma globulin was of value in severe cases.

FELIX ARDEN (Brisbane) considered that any case of pneumonia in a child under 12 months of age should be regarded as staphylococcal.

D. G. HAMILTON (Sydney) favoured the use of digitalis for very sick patients.

Dr. Stuckey, in reply, said that he did not have a record of the use of digitalis in his own cases, and he found the question difficult to answer; but according to the records, its timely use was good. Rib resection should not be recommended early, but it was of value when drainage had continued for a long time.

Mitral Valvotomy.

DOUGLAS STUCKEY (Sydney) presented a report of case entitled "Mitral Valvotomy in a Girl, Aged Seven Years" (see page 812).

MOSTYN POWELL (Melbourne) referred to six cases of mitral stenosis at the Royal Children's Hospital in Melbourne. He said that he thought the condition extremely rare. None of those patients had undergone surgery, but he thought they would certainly need it later on.

ROBERT SOUTHBY (Melbourne) told of a case described in *The British Heart Journal* of post-mortem findings of mitral stenosis at the age of three years. He asked what the long-range prognosis would be for such a child.

Dr. Stuckey, in reply, said that the prognosis depended on the continuation of rheumatic activity. If that was controlled the prognosis would be fair, but it was very hard to state exactly. The indications for operation were, firstly, severe and increasing illness of the child, in which case the prognosis was short, and secondly, hæmodynamic confirmation of the diagnosis.

R. H. VINES (Sydney) confirmed the deterioration in the child described by Dr. Stuckey. He said that at the second observation, prior to operation, he thought that the child had not many months left to live. Although the results were not what was expected, the operation had left her with definite benefit.

D. G. HAMILTON (Sydney) asked Dr. Stuckey whether one should wait until a child became very ill, and what was the earliest stage to consider operation.

Dr. Stuckey replied that in adults one waited until significant symptoms were present. That also applied in children. If definite symptoms appeared, then operation was indicated. Cardiac catheterization was useful if any doubt existed.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE NEW SOUTH WALES HOSPITALS FOR THE INSANE.[1]

[From the *Australasian Medical Gazette*, July, 1897.]

IN reviewing table 4 of the report on these institutions for 1896, the high death rates from general paralysis of the insane and from pulmonary consumption are perhaps the most striking features. That 50 patients—by far the highest number heretofore registered—should in one year have died from general paralysis of the insane, a disease which thirty years ago was almost unknown in this colony, calls for some comment. This disease, always progressive, and ultimately fatal, would appear to be a product of modern civilisation. First described in 1839 as a distinct affection, it remained a rare disease for some years, and was practically unknown forty years ago except to some alien physicians. Even thirty years ago, though well known in England, it was seldom seen in institutions for the insane in Scotland and Ireland, and more rarely still in American institutions. Now it is common enough in all those places, and is very decidedly increasing in all so called civilised communities. It is distinctly an urban disease. More than two-thirds of all the cases admitted to the hospitals for the insane in this colony are to be found at Callan Park where patients from the Metropolitan district are received, and the areas within which it originates are for the most part those pervaded by telephones and other kindred contrivances. Its causation may be expressed in the words hurry, high pressure and excess. There has always been a subtle connection between pulmonary consumption and insanity, and the percentage of deaths from consumption in the asylums of Great Britain has always been a high one and very much in excess of that in this colony. The percentage, however, is now increasing here, and was greater during the last than in any preceding year. The establishment of up-country hospitals for the insane will permit of patients threatened with pulmonary phthisis being sent away to drier and more suitable climates; but it will probably be advisable, now that the pathology of this affection is better known, to make arrangements for the special isolation in separate buildings within the hospital grounds of all cases of this disease in its most infective form.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

ELECTIONS TO THE UNIVERSITY OF SYDNEY SENATE AND THE N.S.W. BRANCH COUNCIL.

SIR: Dr. Klugman raises an important issue when he states that we frequently have to vote for people who are practically unknown to us when we are electing the B.M.A. Council.

There are several aspects of the elections which on matters of principle might call for discussion. In raising these aspects for your interest, let me hasten to say that I have no axe to grind with the B.M.A. Council in Sydney. So far as I am concerned they are all fine fellows; they never quarrel with me, and I hope I shall not quarrel with them. They seem to do a good job, and they have the bother of going to the meetings which most of us do not find to be of any interest.

Nevertheless, there are two factors which require watching: (a) We neglect our duty in the nomination of candidates, leaving the bother to a select few. (b) Only a limited number of doctors seem to record votes, say 1000. The danger of this is the possibility of cliques' electing their pals. From the appearance of the voting papers sent to me, the evidence is very much in favour of this already being the case. It is possible to define at least two groups, if not three.

So far so good. We like them and we vote accordingly; but is it satisfactory? Suppose a communist group got control?

Yours, etc.,

JOHN A. MCCLUSKIE.

133 Wigram Road,
Glebe, N.S.W.
November 1, 1959.

A NEW DRUG IN RELIEF OF ANGINA PECTORIS.

SIR: I was interested in the letter from Professor Blackburn which was published in the Journal, October 3, 1959, page 501, advising against the use of iproniazid ("Marsilid"). He stated that his views were shared by most cardiologists and psychiatrists. I would take exception to the latter statement. There is little doubt that iproniazid is a toxic drug which needs careful administration. From my reading of the literature, the majority of psychiatrists find this drug has a useful, if somewhat limited, psychiatric application, especially in depression with little or no agitation, but loss of weight. Dally[1] has summarized the indications for the use of this drug very adequately. A recent paper by Dibden[2] stresses the practical value of this drug. Wortis,[3] in a review of psychiatric progress in 1958, mentions iproniazid favourably, but stresses the need for careful adjustment of dosage.

I have used this drug in well over 100 patients during the past year, and I have had no cases of jaundice. Sargant[4] has used it in over 500 patients without evidence of toxic damage to the liver. I have not used the drug in a dosage of above 150 mg. per day. It is doubtful whether the toxic effects are related to the scale of dosage, but it should be noted that one of the cases quoted by Pare and Sandler[5] was having 400 mg. per day. They estimate that the complication of an acute hepatic necrosis following iproniazid is about one in 2000 treated patients and the mortality is 15%. On theoretical considerations, I feel that the concurrent administration of barbiturates and phenothiazone derivatives, especially chlorpromazine, should be limited.

In conclusion, I would say that iproniazid has a definite and useful part to play in psychiatric treatment, and if used carefully, with due regard for the complications, it is not a dangerous drug. The chances of it causing hepatic necrosis must be balanced against the risk of suicide occurring in a depressed patient. Many drugs used in medicine are dangerous; but we should not automatically abandon them on this account, but should seek to become more skilled in their use, always bearing in mind the gravity of the illness we are dealing with.

Yours, etc.,

R. A. PARGITER.

Department of Health Services,
Division of Mental Health and Psychological Clinics,
Hobart.
October 23, 1959.

CHLOROTHIAZIDE: A NEW APPROACH IN THE TREATMENT OF HYPERTENSION.

SIR: In reply to Dr. Seldon's letter on this subject (MED. J. AUST., October 17, 1959), it is of course realized that variations in cardiac output are important in making an estimate of peripheral arterial resistance.

Unfortunately, up to the present time no satisfactory method of measuring cardiac output which can be applied conveniently in ordinary clinical practice has been evolved.

[1] *Brit. med. J., 1958, 1:1338.*
[2] MED. J. AUST., 1959, 2:308.
[3] *Amer. J. Psychiat.*, 1958, 115:579.
[4] Personal communication.
[5] *Lancet*, 1959, 1:283.

Many objections can be raised to the method suggested by Starr *et alii* (1954)[1] in the article to which Dr. Seldon refers.

We are dependent, therefore, on pressure measurements alone in making some assessment of total peripheral resistance in hypertensive patients. My point was simply that an estimate of mean arterial pressure, based largely on diastolic pressure but taking some cognizance of systolic pressure, may give a more reliable assessment than consideration of either systolic or diastolic pressure alone.

Yours, etc.,

175 Macquarie Street, Sydney. DOUGLAS STUCKEY.
October 26, 1959.

PREMEDICATION OF CHILDREN.

SIR: The correspondence in your Journal by Dr. Ian McDonald and Dr. I. R. McDonald has just reached my notice. Having introduced the premedication of children with "Oblivon"-hyoscine by mouth, I would like to comment.

The main point at issue seems to be the incidence of vomiting during induction. It surprises me how this possibility continues to worry some anæsthetists, and I can only assume it is a case of "give a dog a bad name, etc.". Dr. Rendell in her original article stated: "In about a tenth of the cases the induction, always slower than after barbiturate premedication, was delayed by vomiting." This paper was based on only 152 cases, and no precise figures are given. I know that in some of her cases the "Oblivon" elixir was given less than one hour before operation, and all were disturbed by giving an injection of atropine some time after the "Oblivon". Dr. Butler, in the letter quoted by I. R. McDonald, actually writes: "As to the incidence of vomiting I have been more fortunate in having only two cases." In what number of cases is not stated. Dr. Hodges, whom I. R. McDonald also quotes, had not tried the drug at all when he wrote his letter. I can definitely state that in my experience, and that of the five other anæsthetists in the group, vomiting in induction is so rare as to be of no more significance than with any other method. This experience now extends over five years and many thousands of cases. I do assure Dr. I. R. McDonald we should have dropped the technique were this not so, as I am sure would Dr. I. H. McDonald.

The timing of the "Oblivon" premedication is most important, and may account for some anæsthetists' difficulties. We now insist that it is given at least one and a half hours before induction, and two hours is quite satisfactory. This is not a technique in which a hurried dose is given just before the child is sent up to the theatre, the list having been altered or the previous case finished quickly. Obviously it is not suitable in cases who are vomiting anyhow—e.g., those suffering from peritonitis.

The actual method of induction is also important. Ethyl chloride is not suitable, and in my opinion is too dangerous a drug to be used at all. My preference is for a "sleep dose" of thiopentone intravenously, and I do assure those who hesitate to try that an intravenous injection is quite easy in a placid child provided a really sharp, fine needle is used. This is followed by nitrous oxide-oxygen and ether, but the induction must be smooth and the ether introduced gradually. Induction is not long by this method.

It is difficult to join issue with Dr. I. R. McDonald on the matter of a child's fears and recollections, as obviously a man who passes stomach tubes on small children has little appreciation of such points. To those interested, I would point out that I have watched Dr. Hodges at work. His children are given atropine only and are then taken into the theatre, where they are given intravenous thiopentone on the table, the whole operation of guillotining the tonsils and curetting the adenoids being done under thiopentone. The wisdom of this requires a whole paper for discussion. In my original article[2] I have pointed out how children do hate and remember injections. To see children cringe at the sight of a syringe is a sad reflection on modern medicine. Those receiving my technique have no recollection of any injection, and certainly no "vivid and unpleasant memory" of a nitrous oxide induction. This week I anæsthetized a boy, aged seven years, for the sixth time, for a plastic repair of a burned hand. His only comment on seeing me again in the anæsthetic room was: "Are you going to put me to sleep the same as before? If so, I don't mind." I challenge Dr. I. R. McDonald to get a similar comment using his method of premedication and induction. It is just because of its simplicity and safety that I commend this technique.

Yours, etc.,

F. R. GUSTERSON,
Senior Consultant Anæsthetist,
Worthing Group Hospitals.

Worthing Hospital, Sussex, England.
October 26, 1959.

[1] *Circulation*, 1954, 9: 648.
[2] *Lancet*, 1955, 1: 940 (May 7).

SKIN STERILIZATION.

SIR: Following my letter of August 29 (which gave the findings of German research workers, reported at the annual meeting of the German Surgical Society last year, that the skin barrier against microorganisms conferred by daily use of commercial preparations containing hexachlorophene is illusory), several of your readers have inquired why there should be such contradiction between investigators in such a simple matter.

The fact is explained as follows. If the hands are treated with the preparations concerned, and then placed in the usual culture media, after incubation little or no growth of microorganisms is found. But if "Tween-80", a wetting agent, be added to the media, or if blood or tissue fluid be placed on the hands, the usual heavy growth of organisms will be found.

The original article is now available in the library of the New South Wales Branch of the British Medical Association.

Yours, etc.,

235 Macquarie Street, Sydney. V. J. KINSELLA.
October 31, 1959.

Medical Practice.

MEDICAL AND ASSOCIATE PROFESSIONS SUPERANNUATION PLAN.

THE following reports were presented at the second annual general meeting of members of the Medical and Associate Professions Superannuation Plan, held at B.M.A. House, 88 L'Estrange Terrace, Kelvin Grove, Brisbane, W.1, on September 8, 1959.

Report of the Founders.

It is with considerable pleasure that I accept the Trustees' Report, which is set out in detail and covers particulars of Plan operations for the period under review.

In such report there is reference to Dr. Robert Miller, who does not, for the present, desire to offer himself for reelection as a trustee. I desire to place on record the valued services given by him over the past three years, in assisting in the creation of the Plan, and with my co-founders, we wish to thank him and express our hopes that he will rejoin the "team" when circumstances permit.

During the year the founders recommended to the trustees, the formation of a holding company in order to add efficiency to the Plan's operations and the registration of M.A.P. Nominees Pty. Ltd. was effected on April 15, 1959.

I believe that the progress made since the inception of the Plan is favourable to members' interests, and the results presented and attained are mainly due to the untiring efforts of the trustees of the Plan.

I desire to extend my sincere thanks to colleagues, both founders and trustees, for their continued cooperation and assistance in this project.

On behalf of the Founders,
NORMAN SHERWOOD, Chairman.

Report of the Trustees.

During the period under review membership has increased by 56; 135 (1958) to 191 (1959). A steady number of inquiries from potential members is being received.

Revenue as disclosed in the statement of accounts amounts to £2,132/10/11, which, with bonus, allows a

distribution to the credit of members' accounts at 5% for the year as against 3·16% (1958).

The contingency fund has been increased to £664/16/9.

The cost price of investments held at June 30, 1959, amounted to £59,461/18/5, whilst the market value calculated at that date was £65,704/4/0, an appreciation of £6,242/5/7.

A list, giving particulars of investment held at the end of the financial year, is appended.

Investments number 57 (35 in 1958).

The above details with additional information are set out herein in tabulated form.

On April 15, 1959, M.A.P. Nominees Pty. Ltd. was registered as a holding company for the Plan. This company does not in any way affect the Plan's operations and general business, except to allow the purchase and sale of stock to be made in the name of M.A.P. Nominees Pty. Ltd. as against the previous procedure of using the name of individual trustees. This overcomes legal difficulties etc., and associated costs which would have occurred in the event of retirement of a Trustee.

On behalf of the Trustees,

J. G. WAGNER, Chairman.

TABULATED DETAILS.

At June 30, 1959.	1958.	1959.
Membership	135	191
Contributions received (excluding life assurance)	£27,238 8 2	£33,957 5 4
Expenses	£236 13 10	£619 13 9
Net profit	£297 17 10	£1,786 14 4
Contributors' benefits (shown on percentage basis)	3·16	5
Contingency fund	£15 0 0	£664 16 9
Membership—Life assurance	61	96
Market value of investment	£22,664 7 6 (at cost)	£65,704 4 0
Appreciation on cost:		
Amount	—	£6,242 5 7
Percentage	—	10·5
Number of investments	35	57
New issues allotted and applied for:		
Shares	—	2651
Notes	—	45

Notes and News.

First Congress of the Asia-Pacific Academy of Ophthalmology.

The first Congress of the Asia-Pacific Academy of Ophthalmology will be held in Manila, Philippines, from October 10 to 13, 1960, under the sponsorship of the Philippine Ophthalmological Society. It will be attended by delegates from different countries of the Asia-Pacific regions, and also by guests from the Americas and Europe. Its main theme will be "Blinding Diseases of the Asia-Pacific Regions". The Committee on Scientific Programme has set February, 1960, as the deadline for the submission of titles of free papers (with brief abstracts of 200 words) and medical films, so that these can be included in the official programme of the Congress. In addition to these papers and films, there will also be guest lectures by prominent ophthalmologists from America, Australia, Europe and India.

Further particulars may be obtained from Dr. Jesus V. Tamesis, Executive Chairman, 42 Quezon Blvd., Quezon City, Philippines.

"Panminerva Medica."

The Italian Medical Association has produced a monthly English language review to be known as *Panminerva Medica*. The first issue, that of May, 1959, celebrates the fiftieth anniversary of the well known Italian journal *Minerva Medica*. It is attractively produced and contains a series of articles representing Italian contributions to many branches of medical science. Comparatively few people in English-speaking countries can read Italian fluently, so *Panminerva Medica* should be a most effective means of bringing Italian medicine to the English-speaking world. It is published by Edizioni Minerva Medica, Corso Bramante 83-85, Torino, Italy. The subscription rate for the twelve issues in the year is £3 10s. (sterling).

Congress of the International Society for the Welfare of Cripples.

The eighth World Congress of the International Society for the Welfare of Cripples will be held from August 28 to September 2, 1960, at New York. The theme of the Congress will be "Rehabilitation and World Peace". The Australian Advisory Council for the Physically Handicapped has been asked to forward the names of Australians who would be competent to take part in the panel sessions; the speakers should be able to talk informatively for fifteen minutes on rehabilitation progress and needs in Australia. The Council draws attention to the need for speakers to be nominated without delay; otherwise the programme will be completed, and there will be no place for late comers. Registration forms may be obtained by those wishing to attend the Congress from the offices of the State Branches of the British Medical Association.

The Eli Lilly Educational Fellowship Programme.

The Eli Lilly Educational Fellowship Programme, which sponsors doctors from all parts of the free world who wish to travel to the United States to further their medical studies, was established soon after World War II. The aim of the programme is to promote a better understanding and establish closer ties between members of the medical profession in the United States and in other countries, and to contribute to post-graduate medical education.

Doctors are selected for fellowships by fellow members of the medical profession in their home countries. In the United States, they study in medical colleges or institutions of their own selection. Their studies may be in general medicine and its related fields or in any of the medical specialties. One hundred and ten fellowships have been granted to physicians from other countries since 1945. The Australian representative among the 1959 International Lilly Fellows is Dr. K. D. G. Edwards of Sydney.

"The Australian Journal of Psychological Research."

We have received the first issue of *The Australian Journal of Psychological Research*. Emanating from the Szondi Clinic in Melbourne, it is to be issued twice a year. Its policy will be to publish contributions on various aspects of clinical, educational and forensic psychology. The annual subscription is 50s., and subscriptions and correspondence should be addressed to the Szondi Clinic, Box 20, 545 St. Kilda Road, Melbourne. The first issue contains four papers on various aspects of homosexuality, three of them by medical practitioners and one by a legal practitioner, and also a case analysis of psychological impotency, by J. Blazsanyik, Ph.D. All three of the mdeical practitioners contributing are shown in the table of contents as possessing the degree of M.D., although the Victorian register of medical practitioners for 1959 shows each of them as possessing only the degrees of M.B., B.S. If this is in fact an error, we hope that it will not be perpetuated in further issues of this new journal. The aim of the journal, which contains book reviews as well as articles, is stated as being (i) to assist in bringing about a heightened spiritual and emotional happiness in the individual, with consequent improved family and community relationships, and (ii) to make available valuable information to people concerned with personal problems and people responsible for care of mind or body or character.

"Biochemical and Biophysical Research Communications."

The first number has been received of a new publication, *Biochemical and Biophysical Research Communications*. It has been initiated, according to an editorial statement in this first issue, to meet the need for the rapid dissemination of information in all areas of experimental biology. This rapid dissemination is the essential aspect of the journal's policy, and an important factor in implementing it is the fact that the articles are not set in printer's type but are reproduced in their original typescript. The editors state that rapid dissemination of new knowledge should prevent unnecessary duplication of effort, and new and exciting information will reach investigators in a minimum length of time. The hope is expressed that all authors will feel free to submit reports of current investigations which will be of immediate value to other scientists and promote the general progress of biochemical and biophysical research. The journal is published by Academic Press Inc., 111 Fifth Avenue, New York 3, New York. It is to be a monthly journal. Volume 1, comprising six issues, is priced at $12.

The Royal Australasian College of Physicians.

EXAMINATIONS FOR MEMBERSHIP: AUSTRALIA, 1960.

THE Royal Australasian College of Physicians announces that examinations for membership of the College will be held in Australia in 1960 as follows: (i) Written examination, capital cities, April 8; clinical examination, Melbourne, commencing on approximately May 18; closing date for applications, March 11. (ii) Written examination, capital cities, August 26; clinical examination, Sydney, commencing on approximately October 5; closing date for applications, July 29.

Applications to appear before the Board of Censors should be made in the prescribed form, and must be in the hands of the Honorary Secretary of the College before the closing date advertised. Candidates should signify in which city they desire to take the written examination. Only those candidates whose answers in the written examination have attained a satisfactory standard will be allowed to proceed to the clinical examination. Application forms are obtainable from the Honorary Secretary, The Royal Australasian College of Physicians, 145 Macquarie Street, Sydney.

College of Pathologists of Australia.

EXAMINATION FOR MEMBERSHIP.

APPLICATIONS to sit for the membership examinations to be conducted during 1960 by the College of Pathologists of Australia should be lodged with the Councillor representing members resident in the State in which the applicant resides. Applications close on February 17, 1960, and must be accompanied by the appropriate fee.

Written examinations will be held in June in the capital cities according to the place of residence of applicants. Viva-voce examinations will be held in Melbourne in August. Full details and application forms may be obtained from the Honorary Secretary, 135 Macquarie Street, Sydney.

Post-Graduate Work.

BULLETIN OF THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that, owing to increased costs, it has been found necessary to increase the annual subscription to its monthly *Bulletin* to £2 2s. The increase will apply from November, 1959. The *Bulletin* consists of lectures given in the annual subscription course and other courses. Further particulars may be obtained on application to the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney".

Public Health.

NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL.

THE forty-seventh session of the National Health and Medical Research Council was held at Adelaide on May 21, 1959. The Council adopted the following resolutions:

Resolution 1: The Council takes note of the report of its Industrial Hygiene Committee and suggests that a campaign of education and advice be taken up with the appropriate employers' organizations with a view to reducing hazards due to excessive noise, and ultimately to recommend legislation to implement the advice if necessary.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 24, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	1	1(1)	..	..	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	4	..	4
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	8(7)	4(3)	..	..	2	..	..	14
Diphtheria	..	1(1)	..	..	..	..	..	..	1
Dysentery (Bacillary)	..	1(1)	..	..	..	..	..	..	1
Encephalitis	1	5(5)	..	..	..	..	..	..	6
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	66(21)	32(16)	9(2)	8(7)	3(1)	..	..	..	118
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1	1	..	1(1)	..	1(1)	..	..	4
Ophthalmia	..	..	..	..	3	..	..	..	3
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	1(1)	..	..	..	..	..	..	1
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	18(7)	..	2(2)	..	..	..	..	20
Salmonella Infection	..	..	..	1(1)	..	..	1	..	2
Scarlet Fever	4(1)	18(14)	11(7)	6	..	..	..	..	39
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	1(1)	..	15	..	16
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	33(23)	18(7)	12(8)	4(3)	2(2)	1	1	..	71
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] No case of notifiable disease was reported for A.C.T. for the week ended October 24, 1959.

Resolution 2: (*a*) That X-ray equipment for use on humans be registered subject to its compliance with prescribed standards. (*b*) That this equipment be subject to inspection and that registration be withdrawn at any time should the equipment not comply with the prescribed standards.

Resolution 3: Council resolves that each State and Territory should undertake study of leukæmia by collection of detailed information concerning cases occurring within its jurisdiction. For this purpose the pro-forma being used by the Cancer Council of Victoria might be used as a basis.

Resolution 4: That the National Health and Medical Research Council recommend to the Minister that a member of the Antibiotics Committee or other suitable representative be sent to the Annual Conference on Antibiotics in the United States and that the expenses for same be obtained by the Commonwealth Department of Health or if possible from the sum allotted to Pharmaceutical Benefits.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Nedeljkovic, Miroslav, M.D., 1934 (Univ. Belgrade), (registered in accordance with the provisions of Section 21C (3) of the *Medical Practitioners Act*, 1938-1958), 3 Lord Street, Cabramatta, New South Wales.

Dwyer, Francis Norman, M.B., B.S., 1956 (Univ. Sydney) (subject to Section 17 (3) of the *Medical Practitioners Act*, 1938-1958), 134 Lord Street, Newtown.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Hamilton, Robert Ian Charles, M.B., B.S., 1958 (Univ. Sydney); Cooke, Ian Douglas, M.B., B.S., 1958 (Univ. Sydney); Bradhurst, Peter Graham, M.B., B.S., 1959 (Univ. Sydney); Muller, Kurt, M.D., 1938 (Univ. Vienna), (registered under the provisions of Section 17 (2A) of the *Medical Practitioners Act*, 1938-1958).

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Garner, John Fairfax, M.A., M.B., B.Ch., 1956 (Univ. Cambridge), M.R.C.S., L.R.C.P., 1955, Base Hospital, Ballarat, Victoria.

Notice.

THE CHILDREN'S MEDICAL RESEARCH FOUNDATION OF N.S.W.

THE following is a list of donations to the Children's Medical Research Foundation of N.S.W. received from members of the medical profession in the period November 4 to 11, 1959.

Dr. Lyle Brown (further), Dr. Leon Gries (further), Dr. R. A. G. Holmes (further), Dr. and Mrs. J. A. Gatenby (further), £10 10s.

Dr. B. A. Curtin (further), Dr. Z. Reiner (further), Dr. A. R. Woolnough, Dr. B. N. Beirman, Dr. W. F. Simmons (further), £5 5s.

Dr. E. A. Jackson (further), Dr. G. T. Hunter (further), Dr. S. D. Bray (further), £5.

Dr. and Mrs. Leonard Green (further), £4 4s.

Dr. T. F. Nevell, Dr. G. C. Spence (further), £3 3s.

Dr. P. H. Doyle, £2 2s.

Dr. G. D. Davis (further), £1 1s. 6d.

Dr. D. Szeps (further), £1 1s.

Previously acknowledged: £9645 1s. 4d. Total received to date: £9743 0s. 10d.

Deaths.

THE following death has been announced:

NEAL.—Leo Augustine Neal, on November 17, at Melbourne.

Diary for the Month.

DECEMBER 1.—New South Wales Branch, B.M.A.: Organization and Science Committee 8 p.m. (with Special Groups 8.30 p.m.).
DECEMBER 2.—Western Australian Branch, B.M.A.: Branch Council.
DECEMBER 2.—Victorian Branch, B.M.A.: Branch Meeting.
DECEMBER 2.—Victorian Branch, B.M.A.: Branch Council.
DECEMBER 3.—New South Wales Branch, B.M.A.: Clinical Meeting.
DECEMBER 3.—South Australian Branch, B.M.A.: Council Meeting.
DECEMBER 7.—Victorian Branch, B.M.A.: Executive of Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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